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ARCHIVES OF MEDICINE.

Original Articles.

(*Concluded.*)

THE THERMIC PHENOMENA IN CONTRACTION OF MAMMALIAN MUSCLES.

(Second communication from the Leipzig Physiological Institute.[1])

By ROBT. MEADE SMITH, M.D.,

PROFESSOR OF COMPARATIVE PHYSIOLOGY IN THE UNIVERSITY OF PENNSYLVANIA.

IN my first communication on the heat development of contracting muscle, I showed that in a long tetanus from maximal irritation, the muscle still in connection with the circulation at first rapidly increased in temperature until much warmer than the arterial blood; that the temperature then slowly increased until a maximum was reached, when, from the decreasing muscle temperature and the increasing arterial temperature, the temperatures of muscle and blood even during the tetanus commenced gradually to approach one another. No conclusion, however, was then drawn as to whether the fall of temperature toward the end of the tetanus was attributable to a more rapid loss of heat through the increase in the blood supply, or to a diminished heat production. The method which I have described above permits the alternate arresting and restoring of the blood supply, during the course of a tetanus of several minutes' duration, and therefore, by the variation of the conditions of the experiment, enables the formation of a conclusion as to this point. For if the cessation of increase of temperature during a tetanus is due to the cooling influence of the blood, arrest of circulation should cause the temperature which had become constant, or even commenced to fall, to again commence to rise ; if, however, under these circumstances the temperature does not increase, it must be concluded that heat production in the muscle has been interfered with, the degree being capable of recognition by the comparison of the temperature of the bloodless muscle during and after irritation.

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[1] See du Bois Raymond's *Archiv für Anatomie und Physiologie*, Physiologische Abtheilung, 1881, p. 105.

In the following experiments of this nature, animals were selected which were especially capable of energetic heat production. In the first experiment the circulation was not disturbed during the irritation until the temperature of the muscle ceased to rise ; in the second, the aorta was closed before the muscle temperature reached its maximum. In the first column of figures to the left is noted the temperature of the muscle immediately before the commencement of the irritation, the succession of figures to the right giving the temperature of the muscle during contraction at intervals of one minute. From the slightness of its changes, the thermometer readings of the arterial temperature are only given at the beginning and end of each period of the experiment ; of these four periods, the first three represent contraction, the fourth rest.

The following curve (fig. 1) has been constructed from the figures of the preceding table. The upper heavy curve represents the temperature-changes in the muscle; the lower lighter curve, the temperature of the blood. The intervals on the abscissa line correspond to minutes ; the heavy ordinates represent the blood-pressure in the femoral artery in millimetres of mercury, while the dotted perpendicular lines represent the duration of irritation and of closure of the aorta.

It is to be noticed in the above experiment that before

I.

																Total Change in Temperature.	
	Time after Commencement of Irritation, in Minutes.																
1. Aorta open.		1	2	3	4	5											Tetanus.
Muscle temperature .	37.83	38.39	38.55	38.70	38.76	38.80° C.										+0.97°	
Temperature increase		0.56	0.16	0.15	0.06	0.04° C.											
Temperature of blood	37.70					37.75° C.											
	Time after Commencement of Irritation, in Minutes.															Total Change in Temperature.	
2. Aorta closed.	6	7	8	9													
Muscle temperature .	38.80	38.80	38.80	38.80												0°	
Temperature increase	0.0	0.0	0.0	0.0													
	Time after Commencement of Irritation, in Minutes.															Total Change in Temperature.	
3. Aorta opened.	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24		
Muscle temperature .	38.80	38.55	38.45	38.30	38.18	38.10	38.08	38.04	38.00	38.00	38.00	38.00	38.00	38.00	38.00	—0.80°	
Temperature decrease		—0.25	—0.10	—0.15	—0.12	—0.08	—0.02	—0.04	—0.04	0.0	0.0	0.0	0.0	0.0	0.0		
Temperature of blood	37.70	37.75	37.72											37.70			
	Time after End of Irritation, in Minutes.															Total Change in Temperature.	
4. Aorta open. — No irritation.		1	2	3	4												Rest.
Muscle temperature .	38.0	37.95	37.94	37.92	37.87											—0.13°	
Temperature decrease		—0.05	—0.01	—0.02	—0.05												
Temperature of blood	37.70				37.70												

the commencement of irritation the temperature of the muscle was 0.13° C. warmer than the arterial blood and that in the first four minutes of contraction this difference was increased to 1.05° C. As the temperature of the muscle then remained constant for thirty seconds, in the presence of the circulation, it was to be expected that if the circulation was arrested, and so one source of cooling removed,

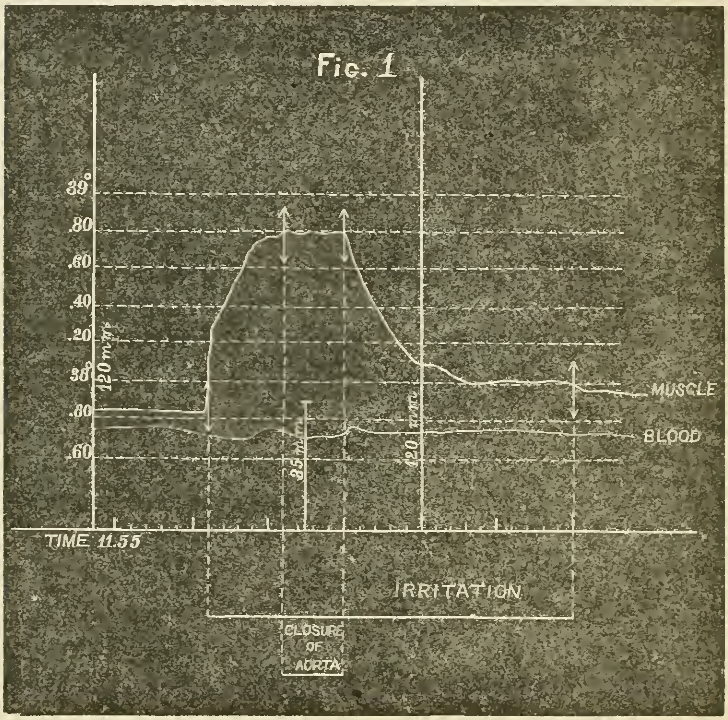

Fig. 1.

the temperature of the muscle would again ascend. Such was not, however, the case: the muscle temperature remained constant during the four minutes that the aorta was closed, and then rapidly sank, when the blood current was restored, to within 0.28° C. of its original temperature, where it remained unaltered until the end of the contraction and then slowly sank to its original stand.

II.

	Time after Commencement of Irritation, in Minutes.										Total Change in Temperature.	
		1	2	2½								Tetanus
1. Aorta open.												
Muscle temperature . .	38.37	38.85	39.07	39.23° C.							+ 0.86° C.	
Temperature increase .		0.48	0.22	0.16° C.								
Temperature of blood .	38.08			38.08° C.								
	Time after Commencement of Irritation, in Minutes.										Total Change in Temperature.	
	3	4	5	6	7							
2. Aorta closed.												
Muscle temperature . .	39.23	39.26	39.27	39.28	39.30						+ 0.07	
Temperature increase .		0.03	0.01	0.01	0.02							
	Time after Commencement of Irritation, in Minutes.										Total Change in Temperature.	
	8	9	10	11	12	13	14					
3. Aorta opened.												
Muscle temperature . .	39.30	39.15	38.84	38.83	38.83	38.77	38.74				— 0.56°	
Temperature decrease .		— 0.15	— 0.31	— 0.01	0.0	— 0.06	— 0.03					
Temperature of blood .	37.95	38.21					38.17					
	Time after End of Irritation, in Minutes.										Total Change in Temperature.	
		1	2	3	4	5	6	7	8	9		Rest
4. Aorta open.—No irritation.												
Muscle temperature . .	38.74	38.56	38.45	38.36	38.29	38.29	38.29	38.29	38.29	38.29	— 0.45	
Temperature decrease .		— 0.18	— 0.11	— 0.09	— 0.07	0.0	0.0	0.0	0.0	0.0		
Temperature of blood .	38.21			38.18			38.15					

The same explanations apply to the following curve, constructed from the above table, as were given for fig. 1.

The above experiment differs from No. I. of the same series in that in this instance the aorta was closed two and a half minutes, instead of five minutes, after the commencement of the irritation, and consequently before the maximum temperature was reached by the muscle; it was therefore to be expected that the temperature would continue to rise after the arrest of the circulation, and it was found that while the muscle increased from 0.29° C. above the temperature of the blood to 1.15° C. in the first period of the experiment, there was a still further gradual addition during the first four minutes, when the aorta was closed. When, however, the circulation was reopened, the muscle temperature fell in two minutes to within 0.63° C. of the

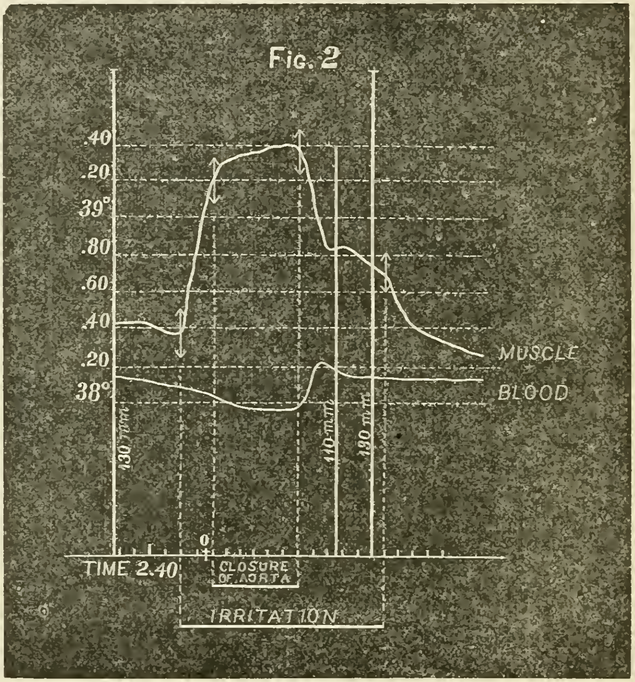

Fig. 2, *a*.

blood temperature, remained nearly constant at this point for two minutes, and then gradually sank still farther during the remainder of the tetanus. When the irritation ceased, the muscle temperature then sank to within 0.14° C. of that of the blood, or 0.15° C. lower than its original position.

By examination of fig. 1 it will be seen that the blood-pressure in the femoral arteries, which at the commencement of the closure of the aorta stood at 0, rose to 30 mm. mercury before the aorta was reopened, from the causes already mentioned in the commencement of this communication as explaining that occurrence. But from the fact that the general course of the experiment corresponds very closely with that of the second experiment, where the closure of the aorta remained complete, it must be concluded that the current in the first case was too feeble to influence the thermic conditions of the muscle.

It may therefore be concluded that the temperature-changes of a muscle in connection with the circulation and contracting under the influence of maximal irritation are dependent mainly upon changes in the heat production of that muscle; and that the heat production which is most intense at the commencement of contractions, gradually decreases in energy until almost entirely suspended. Another fact in favor of this explanation is that during the period in which the temperature-increase commences to decline, the circulation through the muscle remains nearly constant; therefore, since the cessation of increase of temperature cannot be attributed to an increase in loss of heat through an increased circulation, it must be due to diminished heat production. And since we have found that after the arrest of circulation there is no further increase in the temperature of the muscle, diminished heat-production must also be concerned in the fall of temperature which occurs toward the end of prolonged tetani, even when the circulation has not been interfered with.

In order to form a conclusion as to whether the presence of the arterial blood increases the capability of developing heat, the temperature-changes of the normal and bloodless muscles, thrown into contraction by maximal irritation, must be compared. From what has already been stated, it is evident that an excess of heat production in the normal muscle[1] over that produced by the muscle removed from its blood-supply, can only be admitted when the degree and duration of temperature-increase in the first, under unfavorable conditions, largely exceed those developed under more favorable conditions in the latter. Accordingly, from the fact that the results of fatigue are not completely removable by short periods of repose, in the comparison of the degrees of heat production of the bloodless and normal muscle the contraction of the bloodless muscle must precede that of the muscle still supplied with blood. If under such conditions the normal muscle acquires a higher temperature and maintains it longer than the bloodless muscle, the presence of the circulation must aid the development of a larger amount of heat, for the muscle still within the circulation is more fatigued and subject to greater loss of heat than the bloodless muscle.

Of the facts brought out in my experiments made after the above plan, I will first give the comparisons of the maximum temperature-increase produced by irritation of the bloodless and normal muscle.

No. I.—1st. Tetanus. *Without circulation.* Temperature increased from 39.25° to 39.63° C. Maximum = 0.38° C.

2d. Tetanus. *With circulation.* Temperature increased from 39.46° to 39.96° C. Maximum = 0.50° C. Muscle temperature 0.49° C. over blood.

3d. Tetanus. *Without circulation.* Temperature increased from 39.65° to 39.95° C. Maximum = 0.30° C.

4th. Tetanus. *With circulation.* Temperature increased from

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[1] This term will be repeatedly employed to designate a muscle whose circulation has not been interfered with.

39.75° to 40.12° C. Maximum = 0.37° C. Muscle temperature 0.40° C. over blood.

No. II.—1st Tetanus. *Without circulation.* Temperature increased from 34.59° to 35.11° C. Maximum = 0.52° C.

2d. Tetanus. *With circulation.* Temperature increased from 34.59° to 35.27° C. Maximum = 0.68° C. Muscle temperature 0.80° C. over blood.

No. III.—1st. Tetanus. *Without circulation.* Temperature increased from 38.27° to 38.68° C. Maximum = 0.41° C.

2d. Tetanus. *With circulation.* Temperature increased from 38.08° to 38.84° C. Maximum = 0.76° C. Muscle temperature 0.83° C. over blood.

No. IV.—1st. Tetanus. *Without circulation.* Temperature increased from 32.75° to 33.13° C. Maximum = 0.38° C.

2d. Tetanus. *With circulation.* Temperature increased from 32.10° to 32.54° C. Maximum = 0.44° C. Muscle temperature 1.07° C. over blood.

No. V.—1st. Tetanus. *Without circulation.* Temperature increased from 39.26° to 39.72° C. Maximum = 0.46° C.

2d. Tetanus. *With circulation.* Temperature increased from 39.10° to 39.50° C. Maximum = 0.40° C. Muscle temperature 0.32° C. over blood.

Taking an average from the above figures, we find that for the fresh bloodless muscle, protected from loss of heat through the blood, the maximum increase of temperature as a mean was 0.41° C., while for the muscle which entered contraction while already in a condition of fatigue, and through which blood was circulating, which, as an average, was 0.65° cooler than the temperature reached by the muscle, the average maximum increase of temperature was 0.53° C. The share of the blood in the heat development by the muscles is still further emphasized when it is noticed that, with a single exception, in all the above experiments the increase of temperature was much greater in the case of the fatigued muscles which still had their normal blood supply, than in the fresh bloodless muscles; and in the one exceptional case the temperature of the fresh bloodless muscle was only 0.06° C. higher than the more fatigued muscle which still had its blood supply.

In order to form any idea as to the manner in which the blood influences the temperature-changes in contracting muscle, a distinction must be drawn between the characters of the circulation in different periods of contraction. For, at the onset of contraction the circulation through the muscle, as Gaskell has shown,[1] is slackened to such an extent that the conditions are very much the same in the muscles with normal blood supply, and in those whose circulation has been arrested by closure of the aorta, rendering it probable that in the early stages of contraction the development of heat is equal in both instances; since even in the normal muscle the heat development occurs so rapidly that the changes occurring in the interior of the muscle are at first uninfluenced by the sluggish blood-stream. The following table gives the result of my experiments on the influence of the blood current on heat production in the later stages of contraction. The first column of temperature-figures to the left gives the temperature of the muscle immediately before the commencement of contraction, the following figures giving the increase in temperature at every subsequent thirty seconds.

From the commencement of the tetanic irritation until the thirtieth second the temperature increases in the normal and bloodless muscles with nearly equal rapidity; in the subsequent periods of the tetanus, however, we find a marked difference. With the exception of a single case we find that in the bloodless muscle the maximum temperature-increase is always attained within one and a half to three minutes, while in the muscles still within the circulation the temperature continues to increase for two and a half to four and a half minutes. From the fact, therefore, that the normal contracting muscle is able to produce heat for a longer period than the bloodless muscle, it is evident that the blood enables the normal muscle to prolong the heat-

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[1] "Arbeiten des Leipziger physiolog. Instituts," 1877.

Time after commenc'm'nt of contract'n.		30"	1 min.	1½ min.	2 min.	2½ min.	3 min.	3½ min.	4 min.	4½ min.	5 min.	5½ min.	6 min.	6½ min.	
I.															
1st. Tetanus	39.25°	+ 0.22	+ 0.14	+ 0.02	0.00										Without circulation.
2d. Tetanus	39.46°	+ 0.27	+ 0.14	+ 0.08	+ 0.02	+ 0.0									With circulation.
3d. Tetanus	39.65°	+ 0.22	+ 0.06	+ 0.02	0.00										Without circulation.
II.															
1st. Tetanus	34.59°	+ 0.14	?	+ 0.05	+ 0.04	+ 0.04	+ 0.06	+ 0.02	+ 0.03	+ 0.04	?	+ 0.05	?	+ 0.05	Without circulation.
2d. Tetanus	34.39°	+ 0.25	+ 0.11	+ 0.12	+ 0.08	+ 0.06	+ 0.04	+ 0.01	0.						With circulation.
3d. Tetanus	34.50°	+ 0.20	+ 0.08	+ 0.02	+ 0.02	0.0									Without circulation.
III.															
1st. Tetanus	37.83°	+ 0.46	+ 0.10	+ 0.06	+ 0.10	+ 0.06	+ 0.09	+ 0.05	+ 0.01	+ 0.03	0.0				With circulation.
2d. Tetanus	38.27°	+ 0.24	+ 0.06	+ 0.02	+ 0.01	+ 0.03									Without circulation.
3d. Tetanus	38.08°	+ 0.22	+ 0.24	+ 0.16	+ 0.08	+ 0.05	+ 0.01	0.0							With circulation.
4th. Tetanus	38.27°	+ 0.20	+ 0.10	+ 0.05	+ 0.03										Without circulation.
IV.															
1st. Tetanus	39.38°	?	+ 0.32	+ 0.18	+ 0.02	+ 0.11	+ 0.06	+ 0.03	0.0						With circulation.
2d. Tetanus	39.64°	+ 0.23	+ 0.04	+ 0.02	+ 0.01	+ 0.01	+ 0.03	0.0							Without circulation.
3d. Tetanus	39.27°	+ 0.28	+ 0.09	+ 0.13	+ 0.14	+ 0.09	+ 0.07	+ 0.03	+ 0.01	0.0					With circulation.

producing changes which at first are of equal intensity in both bloodless and normal muscles.

We see, therefore, that the blood not only in the periods of rest, but also during tetanic contraction, modifies the thermic phenomena in muscles.

That the above rule is not without exceptions is seen in the first tetanus of No. II. of the above series. There the temperature in the bloodless muscle continued to increase for six and a half minutes, though this tetanus differed from the other tetani in the same animal and in the others of the same series by the extreme slowness with which the maximum temperature was attained; since in the six and a half minutes during which the increase in temperature continued the maximum reached was only 0.52° C. as contrasted with an increase of 0.62° C. attained in the following tetanus of only three and a half minutes, when the muscles were still within the circulation.

If the changes leading to a development of heat in the contraction of bloodless muscles are hindered by the absence of oxygenated blood, it is conceivable that the reopening of the circulation during a tetanus of bloodless muscles after the increase of temperature had ceased might again furnish the necessary conditions for the further development of heat in such a degree as to cause a second increase of temperature. Such a state of affairs occurred in several of my experiments, as seen in the following table.

		Muscle Temperature.	Temperature-Increase in each Minute.	Total Increase.
I.	Aorta closed.	34.34° C.	+0.18, +0.02, +0.01, 0.00,	+0.21° C.
	Aorta opened.		+0.05, +0.08, +0.04, +0.04, 0.04,	+0.23° C.
		Muscle temperature +0.68° C. over blood.		
II.	Aorta closed.	38.27° C.	+0.30, +0.08, +0.0,	+0.38° C.
	Aorta opened.		+0.07, +0.13, +0.08, +0.03,	+0.31° C.
		Muscle temperature +0.77° C. over blood.		
III.	Aorta closed.	36.21° C.	+0.24, +0.0,	+0.24° C.
	Aorta opened.		+0.08, +0.13, +0.03, +0.01, 0.00,	+0.25° C.
		Muscle temperature +0.70° C. over blood.		

The following curve, constructed from the figures of the third tetani of the above series, serves to make the results of the above experiments more evident; the same explanations apply as in the case of fig. 1.

The above experiments, which are confirmed by several others, greatly assist in the explanation of the share of the blood in the internal changes in contracting muscles. For the chemical processes, which in contracting bloodless muscles have sufficient energy to develop enough heat to maintain the muscle temperature from 0.2° to 0.4° above that of the surrounding media, are so increased in energy with the restoration of the circulation that the muscles still further increase in temperature; in spite of the loss of heat attendant upon the circulation of a blood current which may be from 0.68° to 0.77° C. cooler than the muscle.

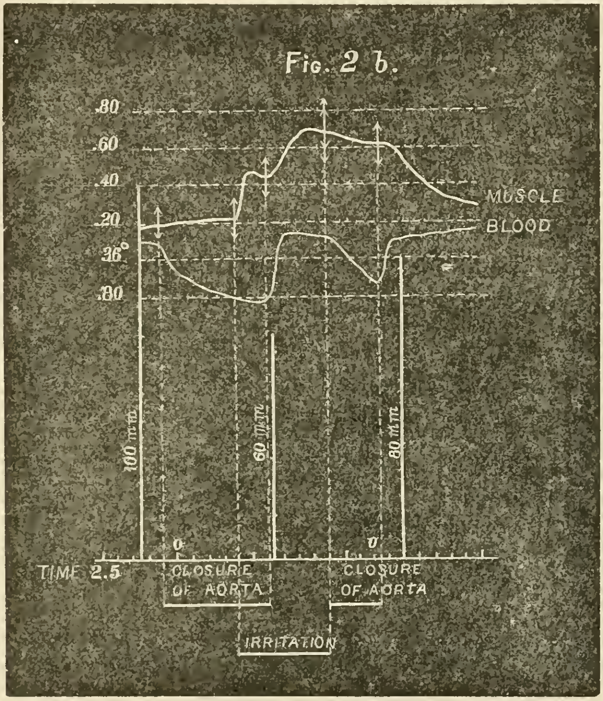

Fig. 2, *b*.

Although I have notes of several other experiments which give the same general results as detailed above, the reopening of the circulation in bloodless muscles during the course of a contraction does not invariably produce such an increase of temperature as in the examples given above. Sometimes the temperature only increases a few hundredths of a degree, and often the cooling influence preponderates over the assistance to heat production; the latter is almost invariably the case when the contraction has lasted more than six minutes before the circulation is reopened.

In the cases which have as yet been considered the muscles which were subjected to irritation, after being deprived of their blood supply, were, during the greater part of the long intervals of repose which followed each contraction, again receiving their normal blood supply. And, although at the end of the contraction the temperature of such muscle was not as greatly increased as after contraction with normal blood supply, the muscle temperature of the bloodless muscle was, nevertheless, considerably above its original temperature and that of its surrounding media.

The following modifications of the plan of procedure were made to permit of the study of the influence which the blood-current exerts on resting muscle after being fatigued and warmed by previous contraction.

The muscles, either with or without their normal blood supply, were first subjected to prolonged irritation; coincident with the cessation of irritation, the aorta was closed (if the circulation had not been already arrested, or was not restored if already closed) and the muscles, deprived of their blood-supply, allowed to rest for ten or fifteen minutes, and then again thrown into contraction without renewing the circulation. Therefore, the muscles were compelled to recover from their fatigue and again contract without receiving any assistance from the arterial blood. These conditions, therefore, reduced the mammalian muscles to the state which alone exists in excised frog's muscles.

Experiments made on the above plan, showed that the muscles, after irritation and an interval of rest while removed from the circulation, were again able to develop heat when again thrown into contraction, but to a considerably less degree than when, after contracting in the bloodless condition, they were a second time thrown into tetanus after an interval of rest in which the circulation was present.

The following experiments show that in the resting muscle contact with the arterial blood facilitates the development of heat in contraction.

I.	Initial Temperature.	Maximum Increase.
1st Tetanus.—The muscles had rested for half an hour with normal blood supply. The aorta was closed at commencement of irritation .	39.26° C.	0.46° C.
2d Tetanus.—The aorta was kept closed during an interval of rest of ten minutes after the first tetanus and during the second tetanus .	39.46° C.	0.08° C.
3d Tetanus.—The aorta was opened after the end of the second tetanus, and the circulation was undisturbed during a pause of ten minutes after the second tetanus, and during the third tetanus	39.10° C.	0.40° C.

Muscle temperature + 0.30° C. over blood.

The course of the above experiment is reproduced in the following curve (fig. 2, *c*):

II.	Initial Temperature.	Maximum Increase.
1st Tetanus.—Duration = 7 minutes. During the tetanus and during a pause of ten minutes before the tetanus the aorta was open .	39.44° C.	+ 1.00° C.
Muscle temperature + 1.00° C. over blood.		
2d Tetanus.—Duration = 5 minutes. The aorta was closed immediately after the end of first tetanus, and kept closed during a pause of fifteen [1] minutes, and during second tetanus	39.97° C.	+ 0.23° C.
3d Tetanus.—Duration = 5 minutes. The aorta was opened immediately after end of		

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[1] During the long closure of the aorta, the circulation was not all the time completely arrested; the pressure in the crural artery, however, during this time, never rose above 10 mm. Hg.

second tetanus and kept open during the subsequent pause of fifteen minutes, and during the third tetanus . . . . . 39.48° C. + 0.74° C.

Muscle temperature + 0.75° C. over blood.

4th Tetanus.[1]—Duration = 6 minutes. The aorta was closed immediately after the end of the third tetanus and kept closed during the subsequent pause of twenty minutes, and during the fourth tetanus . . . . . 39.87° C. + 0.34° C.

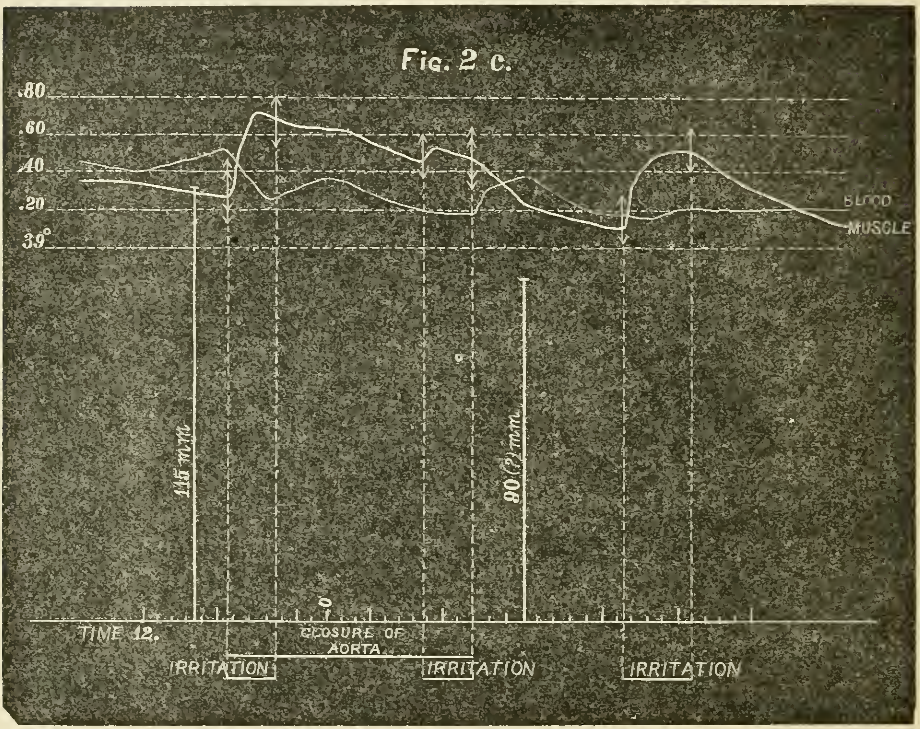

FIG. 2, *c*.

5th Tetanus.—Duration = 9 minutes. Immediately after the end of the fourth tetanus the aorta was opened and kept open during the subsequent pause of twenty minutes, and during the fifth tetanus . . . . . 39.33° C. + 0.85° C.

Muscle temperature + 0.60° C. over blood.

III.

1st Tetanus.—Duration = 5 minutes. Aorta open during the course of this tetanus and for the preceding ten minutes' rest . . . . 38.35° C. + 0.80° C. (?)

Muscle temperature + 0.60° C. over blood.

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[1] Up to this time sub-maximal irritation had been employed ; the strength of the current was now increased up to a maximal irritation.

2*d Tetanus.*—Duration = 5 minutes. Aorta was closed immediately after end of first tetanus and kept closed during the subsequent pause of ten minutes, and during second tetanus .	38.71° C.	+ 0.1° C.
3*d Tetanus.*—Duration = 5 minutes. The aorta was opened immediately after the end of the second tetanus and kept open during the subsequent pause of ten minutes, and during the third tetanus	38.22° C.	+ 0.53° C.

Muscle temperature + 0.53° C. over blood.

It is seen from the above that the resting bloodless muscle can recover from its inability of developing heat, but that this recovery is much more imperfect than when the circulation is present, and that the rapid initial rise of temperature is also in such cases absent. The excess of heat production, therefore, in a muscle which is supplied with blood during its period of rest, and deprived of it during its contraction, over that of the muscle removed from the circulation in both these periods, proves that the arterial blood plays an important part in the conditions essential to heat production by muscles. For were the amount of heat dependent on the store of combustible matter in the muscle, it is clear that the muscle removed from the circulation must liberate less heat than one whose blood-supply is undisturbed.

If fatigued muscles, in the absence of their blood supply, only slightly recover their capacity for heat production, and if, on the other hand, the normal muscle develops more heat in its contraction than the bloodless muscle, the assumption is warrantable that by reversing the conditions of the experiment the course of the temperature-changes in muscle may be radically changed. Therefore, if a fatigued muscle is deprived of its blood supply during its period of rest, and the circulation restored at the moment of commencement of a second irritation, if the above views are correct, the initial increase of temperature will be much less than in the normal muscle, but it will be more gradual and prolonged. The following experiment was performed to test the truth of these arguments:

	TIME (Minutes).																Total Increase.
1st Tetanus . . . . . The circulation was undisturbed before and during the irritation.	½	1	1½	2	2½	3	3½	4	4½	5	5½						
Muscle temperature, 37.94° .	38.17	38.26	38.35	38.45	38.54	38.61	38.67	38.71	38.73	38.76	38.77						
Increase in each 30″ . .	0.23	0.09	0.09	0.10	0.09	0.07	0.06	0.04	0.02	0.03	0.01						+0.83° C.
The difference in temperature of the muscle over that of the blood increased from —0.01° to +0.80° C.																	
2d Tetanus . . . . . The circulation was arrested immediately after end of first tetanus, and aorta kept closed during fifteen minutes' rest, and then opened at commencement of second tetanus.	½	1	1½	2	2½	3	3½	4	4½	5	5½	6	6½	7	7½	8	
Muscle temperature, 38.17° .	38.16	38.16	38.19	38.21	38.24	38.27	38.31	38.37	38.42	38.49	38.53	38.58	38.61	38.63	38.63	38.63	
Increase in each 30″ . .	0.01	0.0	0.03	0.02	0.03	0.03	0.04	0.06	0.05	0.07	0.04	0.05	0.03	0.02	0.0	0.0	+0.46° C.
The difference in temperature of the muscle over that of the blood increased from +0.17° to +0.70° C.																	
3d Tetanus . . . . . The circulation was undisturbed during a pause of fifteen minutes after the second tetanus and during the third tetanus.	½	1	1½	2	2½	3	3½	4	4½								
Muscle temperature, 38.08° .	38.18	38.28	38.46	38.51	38.54	38.58	38.59	38.60	38.60								
Increase in each 30″ . .	0.10	0.10	0.18	0.05	0.03	0.04	0.01	0.01	0.0								+0.52° C.
The difference in temperature of the muscle over that of the blood increased from +0.13° to +0.54° C.																	

The following curve was constructed from the first and second tetani of the above experiment, in order to render the contrast between the rate of increase of temperature more evident.

It is thus seen that the arterial blood plays an important part both in the formation of the heat-giving substances in resting muscle, and in their combustion in contraction; and that even if, although not yet proven, the difference between the bloodless muscle and the muscle still in connection with the circulation is simply a quantitative one, it is nevertheless too important to be neglected. The restriction, therefore, of study to the bloodless muscle alone would be open to the same objection as the application of conclusions drawn from the study of the tissue-changes during hunger to the general metabolic processes of the body. Perhaps in the future it may be possible to isolate the sub-

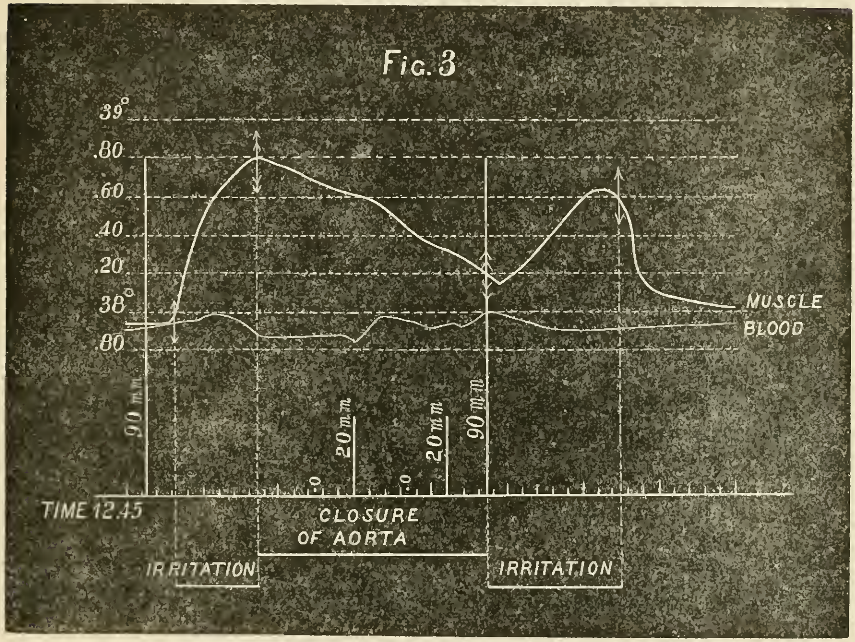


Fig. 3.

stances in muscle through whose combustion heat is developed; but even should this most difficult problem in the chemistry of muscle be solved, measurements of temperature-changes during contraction in the normal muscle, as to their duration and character from the commencement of contraction to the highest degree of fatigue, must still furnish an indispensable and accurate means of studying the course of the process of combustion of those bodies. And, finally, it is only by the study of the temperature-changes in the resting and contracting muscle while still within the circulation, that any idea can be formed as to the share which the muscles play in maintaining the general body temperature; while the comparison of the behavior of the normal and bloodless muscle in this connection is alone sufficient to render indispensable investigations similar to those detailed above.

The Relation between the Work and Heat Production in Contracting Muscles.

The question as to the relation between the work accomplished by the contracting muscle and the amount of heat developed in contraction, must at present be confined to the consideration of the question as to whether under like conditions of irritation and arterial blood-supply the development of heat and change in form vary in direct proportion; for the elaborate and valuable conclusions drawn by Fick as to the laws governing muscular contraction are not applicable to a tetanus which accomplishes no work. To simplify this problem the influence exerted by the blood-current on the course and degree of contraction will be first considered.

If the circulation through the aorta is arrested only a few minutes before or simultaneously with the stimulation of the resting nerve and muscle, a maximal irritation produces a lift which is equal in height to that produced by the normal muscle. But while the energy of contraction is at first equal in both instances, the bloodless muscle rapidly lengthens during the stimulation, so that within one or two minutes after the commencement of irritation the muscle has regained its original length, even though there has been no decrease in the strength of the current applied to the nerve. The following figures illustrate the striking difference between the duration of shortening in the bloodless and normal muscle when subjected to equal stimuli.

a. 1st Tetanus.—*Circulation arrested.* Lift at commencement of irritation = 140 mm.
After tetanus of one minute's duration: lift = 11 mm ; or, as 100 : 7.

2d. Tetanus.—*Circulation normal.* Lift at commencement of irritation = 140 mm.
After tetanus of seven minutes' duration : lift = 121 mm.; or, as 100 : 86.

b. 1st. Tetanus.—*Circulation arrested.* Lift at commencement of irritation = 145 mm.
After tetanus of five minutes' duration : lift = 55 mm.; or, as 100 : 38.

2d. Tetanus.—*Circulation normal.* Lift at commencement of irritation = 140 mm.
After tetanus of five minutes' duration : lift = 106 mm.; or, as 100 : 75.

c. 1st. Tetanus.—*Circulation normal.* Lift at commencement of irritation = 125 mm.
After tetanus of three minutes' duration ; lift = 105 mm.; or, as 102 : 84.

2d. Tetanus.—*Circulation arrested.* Lift at commencement of of irritation = 110 mm.
After tetanus of one minute's duration : lift = 0 mm.; or, as 100 : 0.

The favorable influence exerted by the blood current on muscular contractility is also seen in cases where the aorta is first closed and then opened during an irritation. In such cases, if the muscles were not already fatigued by previous contraction, and if the aorta had not been closed more than a few minutes, when the circulation was restored during the stimulation, the muscle which had become almost entirely relaxed again shortened, though the degree of contraction never reached the point first attained at the commencement of the irritation.

If, on the other hand, the muscle had been already repeatedly tetanized, or if the blood supply had been arrested for a number of minutes, with the restoration of circulation there only occurs a very slow and incomplete return of contractility; showing that recovery only slowly occurs on restoring the blood supply to muscles injured by prolonged irritation and deprivation of blood. Finally, when the experiment as described above is performed on muscles highly fatigued by prolonged and repeated irritations, on reopening the circulation there is no return of contractile energy to the muscle, and the weight is not lifted above its height at the end of the period during which the circulation was closed.

If, in the first place, we compare the energies of contraction and heat-production in the stimulation of bloodless muscles, it is seen that in both the changes run in the same direction. At the commencement of irritation the muscle suddenly attains its highest degree of shortening, and simultaneously the heat-production, which is feeble during rest, is suddenly increased. It must be granted that the rapidity with which the thermometer indicates the maximum temperature falls far short of the rapidity with which the muscle shortens; but this does not necessarily indicate a slower development of heat, for even were the heat instantaneously developed, the low conductivity of the muscle mass and of the glass would require time before the increased temperature could be indicated by the thermometer.

Further, the relaxation, which is rapid in the first few minutes, becomes more and more gradual during the prolongation of the stimulation, and completes the analogy to the temperature changes, for the increase of temperature, which at first is rapid, becomes less and less as the stimulation is prolonged.

The following examples are selected from twenty experiments made on eight different dogs, and may be regarded as giving the possible extremes, as in the first example the evidences of fatigue were only slowly developed, while the muscle rapidly lost its contractility in the second.

Time after commencement of contraction in minutes . .		½	1	1½	2	2½	3	3½	4	4½	5	5½	6
I.													
Lift, in mms. .	145		75	65	65	65	65	55	55	52	52	52	52
Muscle temperature . .	39.59°												
Increase in each 30 seconds .		0.14	0.05	0.04	0.04	0.06	0.02	0.03	0.04	0.02	0.03	0.02	0.03
II.													
Lift, in mms. .	141	136	121	51	6	6							
Muscle temperature . .	39.25°												
Increase in each 30 seconds .		0.22	0.14	0.02	0.0	0.0							

The analogy of changes in the capabilities of the irritated muscle of developing heat and of contracting is still further preserved in the fact that, though both results of irritation have ceased to appear in a muscle fatigued by prolonged weak stimulation, when a maximal is then substituted for a sub-maximal irritation both heat development and shortening again reappear. The following examples serve to illustrate this point; both were obtained from the irritation of the same muscle, one during closure of the circulation, and one during normal blood supply. Their comparison serves to show how closely the muscular energy is dependent on the blood supply. The temperature is given for each thirty seconds after commencement of the tetanus:

1st Tetanus.—*Circulation Arrested*:

	Sub-maximal irritation.								Maximal irritation.	
Lift, in mms.	40		10		0			0	75	59
Muscle temperature	39.87°,	39.87,	39.92,	39.95,	39.97,	? 39.98,	? 39.98,	39.97	40.16,	40.21

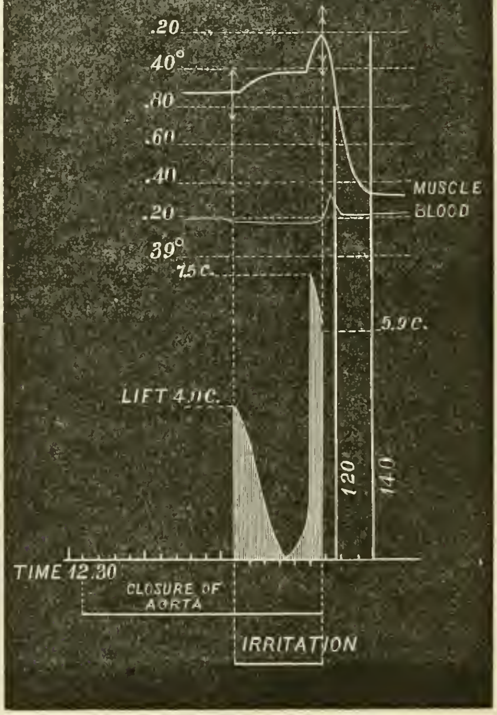
2d Tetanus.—*Circulation Normal*:

	Sub-maximal irritation.												
Lift, in mms.	55		53		47		44				37		30
Muscle temperature	39.33°,	39.40,	39.47,	39.54,	39.60,	39.65,	39.69,	39.72,	39.74,	39.77,	39.79,	30.80,	39.82

	Maximal irritation.			
Lift, in mms.	89			80
Muscle temperature	40.08,	40.22,	40.30,	40.30

The following curves, fig. 4, *a.*, and fig. 4, *b.*, are constructed from tetanus 1 and 2, respectively; the shaded portion of the figure represents the course of the lift.

FIG. 4 A



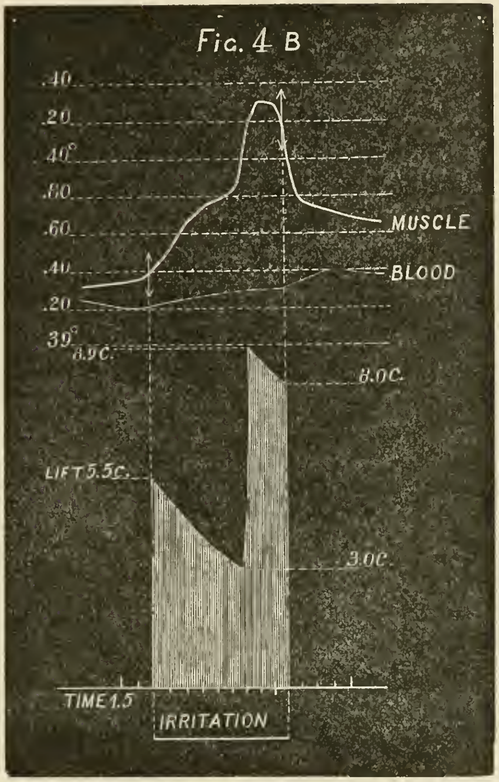


Fig. 4 B

In the muscle which still receives its normal supply of arterial blood the same relation still holds between the energies manifested in the development of heat and accomplishment of work: both forms of energy commence together and disappear together. But it cannot be concluded from this qualitative coincidence that the relation between the liberation of heat and contraction is the same in the bloodless and normal muscle. That with the presence of arterial blood this relation differs from that of the bloodless muscle is suggested by the observations of Heidenhain, which show that in prolonged work the muscle loses its power of developing heat before its contractility is lost; and consequently the invigorating action of the arterial blood supply must produce a different influence on the potential energies of the muscle from that exerted by the fatiguing contraction.

On the solution of this problem depends the most important deduction as to the internal processes in contracting muscle. For if the muscle in accomplishing equal degrees of work can develop unequal amounts of heat, the relation between these two phenomena can be but a limited one, and, as far as I can see, can only be explained in one of two ways: either through alteration in the relative inert mass of the muscle; or from the production by the irritation of two different kinds of internal rearrangement, one manifested by change in form, the other by development of heat.

In the muscle, as in all other machines, the position of the point of application of the force has an important influence on the result. And if, consistently with the conditions of life, changes may take place which permit of a more advantageous application of the force, it is readily conceivable that in spite of a diminished liberation of energy the actual amount of work may remain the same or even increase, as shown by the observations of Heidenhain already alluded to.

The second supposition, that in the irritated muscle two different chemical processes are simultaneously started by the stimulus, and that then each pursues an independent course, though at first sight contradictory, is really conceivable: for the muscle may preserve a condition of contraction, as in the so-called *contracture*, without appreciable development of heat; and, on the other hand, the resting muscle even when deprived of its arterial blood supply may increase in temperature.

The conditions under which the muscles contract in the living animal may be so varied by experiment as to permit of study of the influence of rest and fatigue, both with and without the arterial blood supply, and to lead to the hope that by numerous modifications of the conditions some idea may be formed as to the inter-dependence of heat production and contraction.

To accomplish this comparison of results attained under different conditions, I first subjected the muscle to two successive maximal irritations of one minute's duration; the first instance being in a fresh muscle deprived of its blood supply, the second with the aorta open.

Since we know that the muscle with normal blood supply surpasses the bloodless muscle both in its power of developing heat and of maintaining contraction, it therefore only still remains to be determined in what proportion, under the influence of the blood, the lift and temperature increase. And since in the normal muscle, through partial dissipation through the blood, the amount of heat developed remains unknown, this can only be accomplished through comparison of the increases of temperature in equal intervals of time; and as the degree of contraction in both cases does not lie within the same limits, only a mean between these can be used for comparison, a method which is not open to serious objection where the variations are within such narrow limits as in the example reported.

In the following examples the bloodless muscle was always irritated before the normal muscle: the temperature of the muscle was always above that of the blood.

I.

1st Tetanus.—*Circulation Arrested.*

Temperature increase at end of one minute = 0.36° C.
Lift at commencement of tetanus = 141 mm. } Mean Lift =
" end " = 121 mm. } 131 mm.

2d Tetanus.—*Circulation Normal.*

Temperature increase at end of one minute = 0.41° C.
Lift at commencement of tetanus = 140 mm. } Mean Lift =
" end " = 130 mm. } 135 mm.
Relation of temperature of bloodless and normal muscle = 1:1.14.

Relation of lift of bloodless and normal muscle = 1:1.03.

3d Tetanus.—*Circulation Arrested.*

Temperature increase at end of one minute = 0.28° C.

$$\left.\begin{array}{ll}\text{Lift at commencement of tetanus} & = 138\text{ mm.}\\ \text{“ \quad end \quad “} & = 105\text{ mm.}\end{array}\right\}\begin{array}{c}\text{Mean Lift} =\\ 121\text{ mm.}\end{array}$$

4th Tetanus.—*Circulation Normal.*

Temperature increase at end of one minute = 0.35° C.

$$\left.\begin{array}{ll}\text{Lift at commencement of tetanus} & = 134\text{ mm.}\\ \text{“ \quad end \quad “} & = 111\text{ mm.}\end{array}\right\}\begin{array}{c}\text{Mean Lift} =\\ 122\text{ mm.}\end{array}$$

Relation of temperature of bloodless and normal muscle = 1:1.25.

Relation of lift of bloodless and normal muscle = 1:1.00.

II.

1st Tetanus.—*Circulation Arrested.*

Temperature increase at end of one minute = 0.30° C.

$$\left.\begin{array}{ll}\text{Lift at commencement of tetanus} & = 70\text{ mm.}\\ \text{“ \quad end \quad “} & = 30\text{ mm.}\end{array}\right\}\begin{array}{c}\text{Mean Lift} =\\ 50\text{ mm.}\end{array}$$

2d Tetanus.—*Circulation Normal.*

Temperature increase at end of one minute = 0.46° C.

$$\left.\begin{array}{ll}\text{Lift at commencement of tetanus} & = 59\text{ mm.}\\ \text{“ \quad end \quad “} & = 61\text{ mm.}\end{array}\right\}\begin{array}{c}\text{Mean Lift} =\\ 60\text{ mm.}\end{array}$$

Relation of temperature of bloodless and normal muscle = 1:1.53.

Relation of lift of bloodless and normal muscle = 1:1.20.

III.

1st Tetanus.—*Circulation Arrested.*

Temperature increase at end of one minute = 0.29° C.

$$\left.\begin{array}{ll}\text{Lift at commencement of tetanus} & = 86\text{ mm.}\\ \text{“ \quad end \quad “} & = 55\text{ mm.}\end{array}\right\}\begin{array}{c}\text{Mean Lift} =\\ 70.5\text{ mm.}\end{array}$$

2d Tetanus.—*Circulation Normal.*

Temperature increase at end of one minute = 0.37° C.

$$\left.\begin{array}{ll}\text{Lift at commencement of tetanus} & = 85\text{ mm.}\\ \text{“ \quad end \quad “} & = 82\text{ mm.}\end{array}\right\}\begin{array}{c}\text{Mean Lift} =\\ 83.5\text{ mm.}\end{array}$$

Relation of temperature of bloodless and normal muscle = 1:1.24.

Relation of lift of bloodless and normal muscle = 1:1.18.

If instead of the above quotients of temperature-increase it were possible to estimate the amount of heat developed, there can be no doubt but that it would be found that the excess of heat production in the normal over the bloodless muscle would be even greater than appears in the above experiments, since my earlier experiments showed that even in the first minutes of muscular contraction the venous blood coming from the muscle is warmer than the arterial supply going to it. Nevertheless, in spite of the unfavorable condition of the calculation it is clear that the resting muscle draws from its arterial blood supply a greater heat-producing than contracting energy.

The experiments above detailed, however, do not yet warrant the conclusion that more heat is developed in normal contracting muscle from the combustion of the matter stored up in its interior during its period of rest than would be the case in equal degrees of contraction in the bloodless muscle; they, however, indicate the manner in which, to be conclusive, new experiments must be made.

The relation between the temperature and lift of the muscle is, as a rule, inverse, when, instead of a fresh muscle, one already fatigued by prolonged irritation and deprivation of blood supply is again thrown into contraction after renewal of circulation.

Thus, when the capability of producing heat has been greatly reduced by prolonged irritation and repeated closure and opening of the aorta, the reopening of the circulation often produces a marked increase in the energy of the contraction, as shown by a higher lift, with but slight change in the temperature.

The following experiment and curve (fig. 5) will serve to make this clear. During the nine minutes through which the irritation lasted the circulation was at first arrested, then opened, again closed, and finally again opened.

a. Circulation Arrested.

Time after commencement of contraction		½	1	min.
Muscle temperature	39.14° C	39.37	39.40	
Lift	141	86	11	mm.

b. Circulation Normal.

Time after commencement of irritation	1½	2.	2½	3	3½	4	4½	min.
Blood Temperature	39.12	?	39.29	39.32	39.31	39.30	39.28	
Muscle Temperature	39.43	39.48	39.48	39.44	39.40	39.37	39.37	
Lift	106	109	109	110	110	110		mm.

c. Circulation Arrested.

Time	5	5½	6	6½	7	min.
Muscle Temperature	39.37	39.36	39.39	39.41	39.43	
Lift	81	9	8	9		mm.

d. Circulation Normal.

Time	7½	8	8½	9	min.
Temperature of blood	39.34	39.39	39.37	39.35	
" " muscle	39.44	39.43	39.40	39.40°	
Lift	9	96		106	mm.

One Minute after End of Tetanus.

Temperature of blood	39.40
" " muscle	39.34

Three Minutes after End of Tetanus.

Temperature of blood	39.37
" " muscle	39.35

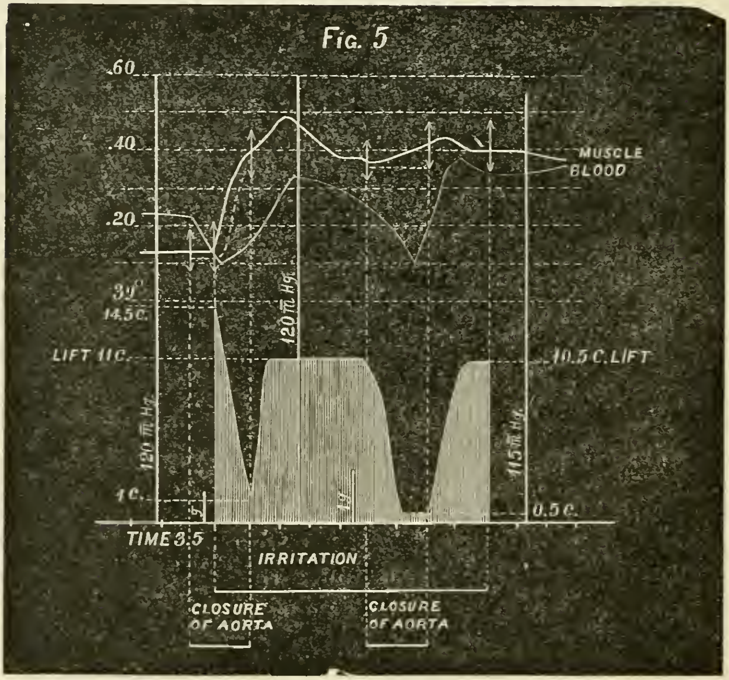


FIG. 5.

In the second period (*b*) of the above experiment the excess of muscle temperature over that of the blood sank in three and a half minutes from + 0.31° C. to + 0.09°, while the lift increased from 106 mm. to 110 mm.; in the fourth period (*d*) the excess of muscle temperature decreased in two minutes from 0.10° C. to 0.05°, while the lift during the same period increased from 9 mm. to 106 mm. In the second period the average decrease in excess of muscle temperature was 0.06° C. for each minute, and in the fourth period 0.025° C., while after the end of irritation it amounted to 0.03° C.

The results of the above experiments correspond with the conclusions drawn by Heidenhain from investigations made on the excised frog's muscle, but the results which were obtained have not been invariably uniform. Among the deviations, the following experiment furnishes an example: Before the tetanus occurred whose course is here described, the muscle had been already subjected to repeated irritations, although immediately preceding the final contraction it had an interval of rest of ten minutes, during which it was supplied with arterial blood for five minutes, and again deprived of blood for five minutes. It was then tetanized for eleven minutes, among the first three of which the aorta remained closed, and was then opened and remained open to the end of the experiment. The following curve represents the course of the experiment.

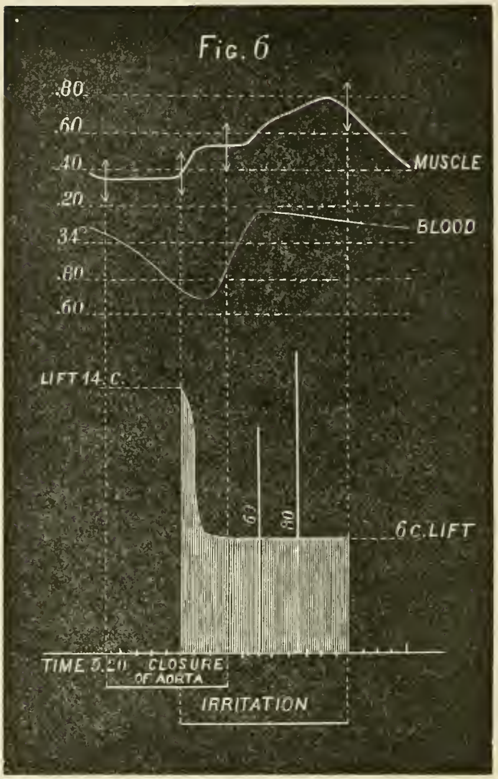


FIG. 6.

In the first portion of the experiment, the result coincides with what had previously been found. When, however, the circulation was restored, no increase occurred in the lift, but nevertheless the temperature, after a slight delay, gradually rose 0.23° C. during the following six minutes, or as great an increase as occurred at the commencement of the irritation.

In conclusion I must regret that circumstances prevented my carrying the above study to its completion, but trust that the results above recorded will assist others in extending a subject which must yield most valuable results as to the relations between the blood and the internal vital processes in muscle, and as to the share of the heat production in muscular contraction in maintaining the bodily temperature.

MULTIPLE NEURITIS.*

By S. G. WEBBER, M.D.,

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NEURITIS has lately assumed an importance which formerly was not accorded it. Local traumatic and rheumatic neuritis have been observed many times, and their symptoms have been well described. The grosser and more marked anatomical changes were well known to the profession years ago. The secondary degenerations which arise in the peripheral ends of divided nerves have been known since Waller described them. More recently various trophic changes in the skin have been correctly referred to disease of the nerves.

It is only within a few years that a more general inflammation of the nerves has been recognized. As has frequently happened, the disease had been seen and cases reported at intervals,—in 1864 by Duménil, Lancereaux in 1870 or 1871, and Eichhorst in 1877,—but attention was not specially attracted to the subject until Joffroy in 1879, Leyden in 1880, and Stewart in 1881, had each described cases. Even then it was some months before the possibility of a general diffused neuritis was fully recognized, if indeed it is as yet. In 1882 Caspari published his article "Zur Casuistik der Neuritiden." In 1883 appeared Pierson's "Uber Polyneuritis acuta" (multiple neuritis), an article by Strümpell, one by Vierordt, and another by Müller in the *Archiv für Psychiatrie*, etc.

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* Read before the American Neurological Association, June, 1884.

Up to this time I have found the records of six cases without autopsies and twelve cases with autopsies—one, however, very brief. As so few cases have been reported, the following reports and summary may be of sufficient interest to claim the attention of this Society.

Case 1.—Thos. McG., æt. twenty-two. Entered Dec. 7, 1883. Was well until one year ago. Since then he has had pains in the abdomen and back, at times lasting several hours, and of a severe darting character. No digestive disturbance. Constipation. Said he had eaten some poorly cooked ham, after which he and others had headache. Nov. 27th he took cold, and Dec. 2d had pains in the legs, and the next day in the arms, a constant aching of the muscles with soreness, worse in the right arm and left leg. Swelling of arms and legs. No pain in back on admission. Chilly and feverish much of the time. Severe headache on Dec. 2d and 3d; dizzy on rising. Vision good; some tinnitus.

On examination it was found that there was tenderness over the biceps on both sides, over the muscles of the right forearm, and over the left radial nerve; not over the other nerves of the arms. Tenderness also over calves and thighs on both sides, over peroneal nerve and sciatics. Both hands and both feet were swollen; no swelling of arms above the wrist, though the patient thought there was. Face was much swollen. There was no special tenderness of the joints, and no swelling of joints, excepting those of the hands and feet, which were included in the general swelling of those parts.

Both arms were contracted in a partially flexed position, the left arm being most contracted. All the muscles below the hips were more or less paralyzed.

Plantar reflex was most marked on the left. Patellar reflex was absent on both sides; no ankle clonus.

Urine was normal in color, acid. Sp. gr. 1025. No albumen; sediment—a large amount of amorphous urates.

Respiratory sounds were normal throughout both lungs.

The faradic reaction was not tested until after the patient had been in hospital several days; then it was found that the tibialis anticus, peronei, and vastus externus on both sides responded feebly even to a strong curve. The rectus femoris and vastus internus did not respond at all. The interossei reacted moderately well; the extensors in forearm, supinator longus and biceps, reacted scarcely at all ; the triceps, deltoid, and flexors responded somewhat better. The facial muscles responded with very nearly the same strength as in health.

The treatment was salicylic acid, iodide of potassium, electricity. The swelling, pain, contraction, and paralysis gradually yielded, and after about two months and a half he could get about fairly well. The œdema reappeared occasionally. The patellar tendon reflex did not return before he was discharged. At times there was profuse perspiration. During the first three weeks the temperature was above normal, once reaching 104.5°. After that the temperature was generally normal. The pulse was almost constantly ninety or above.

April 7th.—He was seen. There was no contracture remaining. No tenderness of legs or arms. There was absence of patellar reflex on the left, it was very slight on the right. He considered himself well and strong.

Case 2.—I. R. æt twenty-eight, entered the hospital, October 26th. She was well and strong until two months ago. She denied syphilis ; had done considerable hard work. Two months ago while at the beach she " took cold " during her catamenia, after which she began to have pain in the abdomen ; this was followed by numbness and loss of sensation. There was a girdle sensation about the waist which later disappeared. No pain in the back. The numbness gradually passed downwards, leaving the abdomen, and on entrance was confined to the knees and legs. Occasionally there were sharp pains in the legs, at other times a dead aching. She could move her legs and feet, but the legs were slightly flexed at the knees, and attempts to extend them or to sit up caused feeling of distress about the heart. She was unable to stand. The pain in the legs was chiefly in the knees, calves, and feet, and was worse at night. There was a slight numbness in the hands. There was no headache, nor dizziness; vision was good, there had been no diplopia ; once a slight tinnitus. Occasionally a slight cough, pain in chest and dyspnœa ; no palpitation. Catamenia were regular, not painful, for last two months delayed three or four days. Appetite and digestion were good, bowels regular, micturition normal.

Nothing abnormal was found in lungs or heart. There was general tenderness over the muscles of the legs and thighs, and in the arms near the wrist, and over the popliteal and sciatic nerves. There was diminution of tactile sensation in the left leg as compared with the right. The plantar reflex was fairly good ; patellar tendon reflex was absent on both sides.

To the faradic current on the right, the tibialis anticus acted fairly well, the peroneus, rectus femoris, vastus internus, and gastrocnemius not quite so well. On the left, the tibialis anticus acted best, the rectus femoris gave scarcely any reaction, the gastrocnemius also responded very sluggishly.

With the galvanic current on the right the tibialis anticus responded readily and gave a quick contraction to fourteen cells; the peroneus gave a slow but strong contraction to twelve cells; the gastrocnemius responded very sluggishly to sixteen cells. Very nearly the same reactions were obtained on the left.

The pain and tenderness increased in the legs and extended to the upper extremities, the nails and ends of the fingers being first affected. There was tenderness over all the larger nerve-trunks in both arms.

At one time the cough had a barking sound and seemed to be somewhat hysterical in character.

The treatment was salicylic acid, ten to twelve grains every three hours; blisters over tender nerves in the arms; morphia to relieve pain; five-per-cent. solution carbolic acid on compresses over painful muscles. This last application gave considerable relief to the pain.

Nov. 16th.—Most of the pain had left the legs and fingers; the fingers were anæsthetic, their ends tender on pressure; pressure to calves also caused pain. The tenderness had disappeared from over the ulnar and radial nerve, was still considerable over the median and popliteal on both sides, slight over the left sciatic.

Nov. 21st.—The pain was entirely gone from the legs and arms, but the legs were still partially flexed and could not be extended without pain.

Jan. 19, 1884.—Tenderness was quite gone from the fingers, except the forefingers. The grasp of the hands was of moderate strength and the same on both sides. The calves of both legs were still tender. The peroneus did not respond to the faradic current on either side. The gastrocnemii and recti femoris required strong currents. The vastus internus did not respond on the left; it required a strong current on the right.

There was contraction of the flexor muscles of the legs, so that the legs were bent at nearly right angles to the thighs; the feet were also in extension and an effort to rectify the position of either the legs or feet caused considerable pain. By means of massage, extension by weights, by elastic bands, and with the aid of electricity the vicious positions were rectified.

Five months after admission she was able to walk with assistance, the heels did not touch the floor in standing, and she could not stand upright, stooping forward. The calves were still tender to pressure, and there was tenderness over the popliteal nerve; patellar tendon reflex was absent.

A month later she was able to walk without assistance and helped in the work of the ward.

Case 3.—Caroline M., æt. thirty-four. Was received Jan. 18, 1884, with a family history of consumption. She was said to have had rheumatism for several years; and for a long time had had palpitation and dyspnœa. Had been somewhat addicted to the use of alcohol. No cough, micturition normal. Cata. regular. She had had an attack of vomiting three months previous to present illness.

In the earlier part of illness, which began about four weeks before entrance, there were pain and numbness in the legs; this improved somewhat, then the pain returned. There was no disturbance of motion excepting as explained by the pain. This account was given from recollection by her physician after her death.

On admission she complained much of pain, chiefly in the joints and back, which had troubled her for three weeks; was unable to turn herself in bed on account of pain; she could not bear to have the ends of the fingers touched. She perspired profusely; was delirious.

Tenderness over forearms, most marked on right; over median nerve on left, median and ulnar nerves on right, and over right clavicle. The calves were also tender, as well as the popliteal and sciatic nerves in legs. Entire paralysis of hands and fingers, legs and feet, and perhaps of thighs. Anæsthesia to pricking and touch in left leg and thighs. Œdema of ankles, feet, legs, and hands. Absence of patellar tendon reflex and plantar reflex. She says that occasionally she chokes on swallowing, but at visit swallowed without difficulty. Voice seemed to be changed but was not nasal. The pain seemed to become less severe; at seven P.M., Jan. 19th, she died. No autopsy.

Case 4.—Mary N., entered the City Hospital Nov. 13th; no special family tendencies; no severe illness. Three years ago had a bubo in groin, and husband had gonorrhœa at the same time. Had two children; no miscarriages; first child died at four months of age; had indulged in excessive use of alcohol, and four weeks before entrance had drunk much, and had a severe attack of vomiting lasting two days, was delirious three or four days, and saw all sorts of things. For some days before entrance, how long is not mentioned in the records, she had slept poorly on account of dull aching pains in lower extremities ; had numbness in hands and forearms. The pain in limbs became severe, she could not turn over in bed without assistance, her knees were drawn up, and attempts to straighten the legs caused severe pain. There was loss of power in extensors of the wrist and hands, and the fingers could not be shut perfectly. There was general tenderness over the muscles of the extremities. Pressure on right over the radial, median, and ulnar nerves, on the left over the median, caused much pain. In the legs pressure was painful over the popliteal, sciatic, and crural nerves on both sides. The voice was hoarse, and she complained of sore throat ; the fauces were of a rather deeper purple tint than normal. Her general appearance indicated so much suffering that a complete examination was not made. Inquiries as to a sore throat previous to admission, and as to the possibility of diphtheria, showed that she had not suffered from any such disease.

Two or three days later it is noted that there was considerable anæsthesia of fingers and feet ; no patellar tendon reflex. The pain was so severe that morphia was given to control it. The paralysis became more complete, the anæsthesia increased, the pain was rather less, or was controlled by the morphia. The voice was reduced to a whisper, and even whispering necessitated great exertion. Urine and fæces passed involuntarily.

Nov. 28th.—There was considerable difficulty in breathing, respiration rapid ; less pain, much thirst ; was said to swallow perfectly well. Air entered the apex of right lung less readily than the left ; an occasional râle was heard ; percussion resonance not so good on right. Respiration was accomplished by means of the upper ribs and the accessory muscles ; there was paralysis of the diaphragm. The respiratory murmur could not be heard lower than the lower edge of the sixth rib. She died seventeen days after entrance.

Autopsy seventy-two hours after death by Dr. Gannett. The lungs showed on section small nodules, grayish and granular, denser than the lung substance. These nodules, about the size of a pin's head, could be felt by pressure from without. On pressure of cut surface, numerous points of pus were to be seen. The bronchial mucous membrane, as well as that of the larynx, trachea, and pharynx, was covered with a puriform fluid. The kidneys were congested, and there was acute purulent pyelo-nephritis. On section of the liver the central parts of the acini were distinct as brownish areas one mm. in diameter ; the peripheral parts were opaque reddish gray. The miscroscope showed central fatty infiltration, pigmentation, and cloudiness of cells of remaining portion.

The brain and spinal cord seemed to be healthy, both externally and on section.

The brachial plexus on both sides with their larger branches as far as the wrist, the sciatics with branches as far as the heel, the crurals as far as the knee, the phrenics, and vagi, were removed.

These various nerves showed nothing abnormal in their gross appearance ; there was no enlargement nor undue redness.

Microscopic Examination.—Numerous sections of the cord were made in various parts, chiefly at the cervical and lumbar enlargement ; more than a hundred were carefully examined. The cells of the anterior cornua showed no variation from the normal in number nor in character ; the white and gray substance seemed to be healthy ; there was no undue injection nor disease of the blood-vessels.

Portions of the nerves were put into a $\frac{2}{3}$-% solution of osmic acid for twenty-four hours, were then placed in a solution of picro-carmine for two days, then placed in glycerine until they could be examined. The parts thus treated were the upper part of both brachial plexuses ; the distal ends of the median, ulnar, and radial on both sides ; the lower end of the longest branch of both sciatic systems, from near the heel ; the right pneumogastric and right recurrent laryngeal ; the small portion of the pneumogastric from near the stomach ; the upper part of the right phrenic. (The rest of the phrenic had been removed with the thoracic viscera, and could not be obtained.) Except the phrenic, and that part of the pneumogastric from near the stomach, and the upper parts of the brachial plexus, every nerve thus examined was much diseased. The lower ends of the sciatic system were most extensively diseased. The recurrent laryngeal and the vagus, just below where it is given off, were least affected. In one brachial plexus, among many specimens examined, one degenerated nerve fibre was found ; in these specimens from the brachial plexuses were found some fibres which seemed to be in the very first stage of inflammation, but most of the fibres were healthy. The distal ends of the median, ulnar, and radial on both sides were much diseased, but less so than the nerves from the leg.

The portions of nerve not placed in the osmic acid were put into Müller's fluid.

The simplest change found was in the medullary sheath at Ranvier's constrictions, without break in the axis, and, so far as I could judge, without increase of nuclei.

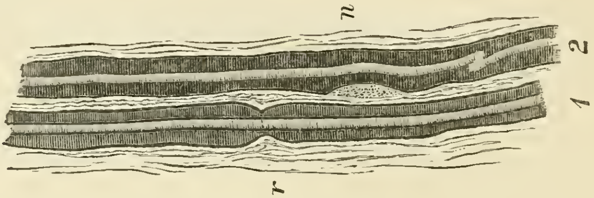

Fig. 1.—Healthy nerve fibres, osmic acid preparation. *r*, constriction of Ranvier; *n*, nucleus. The axis cylinder is bent on itself in one place.

The changes in the distal ends of the nerves were very similar or identical with what is found in secondary degeneration after section of a nerve. The medullary sheath was divided into fragments of greater or less extent; the axis-cylinder was discontinuous or destroyed, remaining when the medullary sheath was only slightly changed. The fragments of the medullary sheath were finally reduced to very small granular débris, and much of this was absorbed. In the fibres which were most altered was found an increase of nuclei; and apparently small masses of protoplasm, not nuclei, were tinted in the midst of the granular débris.

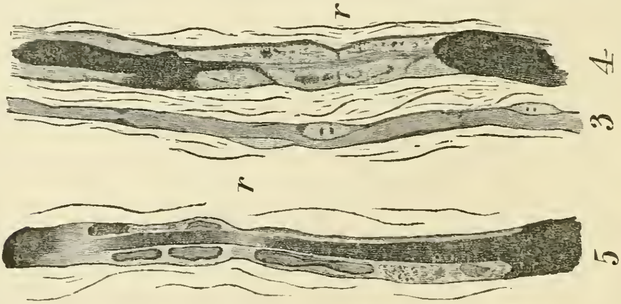

Fig. 2.—At 3 a thin fibre, with three nuclei in or adherent to it; at 4 and 5 inflammatory changes just beginning in the medullary sheath at the annular constrictions *r*.

On cross-section of portions hardened in Müller's fluid and tinted with carmine, the distal ends, especially of the branches of the sciatic, showed almost no nerve fibres with axis-cylinders; the sheath was either filled with granular material or had collapsed. There were many small masses, either of fibres or nuclei, which were tinted almost uniformly a deep red. Nearer the root of the

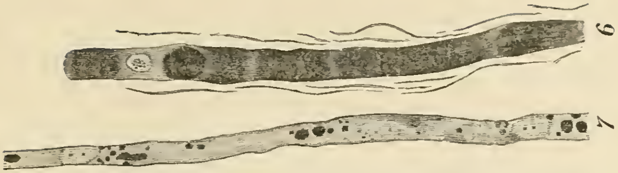

FIG. 3. The first segmentation of secondary degeneration at 6; extreme degree of this degeneration, only a few points or dots of myeline showing, 7.

nerves, axis-cylinders were more numerous, and specimens yet nearer the origin showed a large preponderance of healthy nerve fibres. In the sciatic the sections from the highest part of the nerve which had been preserved were still diseased in a small proportion of their fibres.

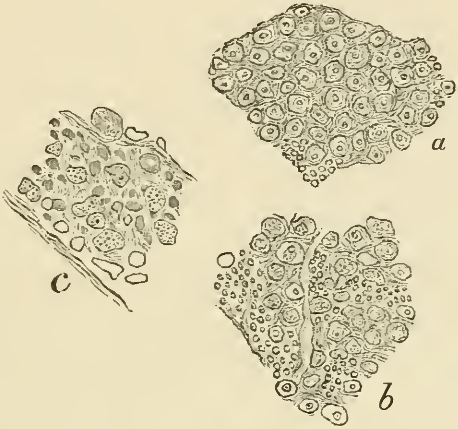

FIG. 4.—Transverse sections of nerve hardened in Müller's fluid, stained with carmine. *a*, nearly healthy from brachial plexus; *b*, much diseased from radial; *c*, extreme degeneration from sciatic.

These few cases are sufficient as examples of this disease. It happened that during the fall and winter of '83–'84, an unusually large number of cases of multiple neuritis entered the City Hospital—eighteen. I saw one in private practice, and a second in which the diagnosis was somewhat doubtful, between neuritis and infantile paralysis. Fourteen of those at the City Hospital came under my observation, nine of these on their entrance, five as remaining when I took the service from Dr. Denny.

Of all the patients the youngest was nine years old (a private patient). This is the only child in the list. The oldest was fifty-one years. More than half were between twenty and thirty years of age.

Where the commencement of the disease could be fixed with any degree of certainty, there was an interval of from one week to four months before the patient entered the hospital. The stay in hospital varied from two days (patient died) to six months ; three or four months in most cases, and even after four months' stay only a few patients left the hospital able to engage in their usual occupations; generally there was still great weakness of the limbs.

In most cases no cause could be given for the disease; in five cases exposure to wet and cold, " catching cold," was thought by the patient to be the cause of his sickness.

Nine were females. Ten were males.

There were three deaths: two of the cases in which death occurred have been reported.

Disturbance of sensation is one of the most constant and prominent symptoms. A tingling or " sleepy " feeling may precede the more severe pain ; within a comparatively short time the pain becomes severe; it is confined to one nerve district or may extend to several; sometimes the patient can mark the course of the nerve by the pain. The character of the pain, aching, boring, shooting, burning, varies in different cases. With the pain there is great hyperæsthesia of the skin and tenderness of the muscles, pressure causing excessive pain. Pressure over the nerve trunks gives rise to more acute pain than when it is applied to the muscles.

When the hyperæsthesia has subsided sufficiently there will be found diminution of tactile and other sensations. The special senses have only rarely been found affected.

Exceptionally the pain and tenderness have been very slight or wanting.

Motion is early disturbed, at first a stiffness, in part due to pain; later paralysis, more or less complete. The muscles are not affected equally, but the paralysis is distributed according to the nerves attacked.

Within a comparatively short time the limbs are contracted, semi-flexed, and sometimes the contraction may become extreme, the heels may be drawn up to the thighs, and the knees nearly to the chest.

The electrical reactions of the muscles and nerves show the changes due to their respective degeneration.

A careful examination of the reflexes, deep and superficial, was made by Dr. Knapp, house officer at the hospital. He found that the various tendon reflexes were generally absent, the patellar tendon reflex being slightly marked in only two out of thirteen patients; the radial and ulnar periosteal reflexes were present in only one or two patients.

The cutaneous reflexes were less disturbed. The plantar reflex was present in eight patients, in two of these rather exaggerated; was absent in five. The cremaster reflex was present in five out of eight males; the abdominal reflex was present in four, absent in nine.

In four other patients not examined by Dr. Knapp, I found the patellar tendon reflex absent; the plantar reflex was also absent in three.

The temperature and pulse were rather elevated in the earlier stages of the disease, and in the two fatal cases throughout the period when they were under observation. Later the temperature dropped nearly or quite to the normal, but the pulse rate continued rather high.

In a few cases a mild delirium was noticed, most marked at night; in one or two women there seemed an hysterical disposition, but neither the delirium nor hysteria were of more than secondary importance.

There was atrophy of the muscles, and in some patients great general wasting of tissue and loss of flesh.

In other patients there was œdema of the limbs and even of the face; this œdema in case 1 affected the hands and feet, in case 3 was more general, and might have led to doubt as to diagnosis, simulating rheumatism.

Excessive sweating was observed in some of the patients, in this respect also simulating rheumatism.

After several weeks' continuance of the disease the skin of the fingers in one patient assumed the glossy appearance peculiar to the trophic disturbance seen in some nerve lesions.

One patient had abscesses in the parotid region during convalescence.

The disease began most frequently in the legs, was sometimes confined to them, but often extended to the arms. In fatal cases its progress was rapid, the respiratory muscles became paralyzed, the respiration was diaphragmatic just before death; the voice was affected. and deglutition was abnormal; the patient died of apnœa, or in case 4, perhaps also from the entrance of food into the lungs. In case 4 death occurred seventeen days after entrance. Case 3 had been sick about four weeks before entrance, though the report is not entirely clear, as she seems to have improved at one time and then became worse again.

Now that the peculiar symptoms of multiple neuritis have been clearly pointed out, the diagnosis is not difficult in well-marked cases, but in light cases might be difficult.

It is to be diagnosticated from anterior poliomyelitis by the pain and hyperæsthesia, the tenderness over nerve trunks, the diminution of sensation, the high pulse rate; by its more gradual onset from the acute form of poliomyelitis; it is less common for the latter to invade all four extremities.

Progressive muscular atrophy does not have the sensory disturbances; the electrical changes are not the same. In lead-paralysis also the sensory disturbances are usually less marked, and the progress is slower.

Multiple neuritis may be mistaken for rheumatic fever, and when there are œdema of the limbs and profuse sweating, as in the first and third cases, there may be reason for making such a mistake. The course of the disease would soon show the mistake. Spinal meningitis will be recognized by the greater amount of pain in the back, by passive motion increasing this pain, by the spasm if present, and by the absence of other peculiarities of neuritis.

Déjérine (Étude sur le névro-tabes périphérique—ataxie locomotrice par névrites périphériques, avec intégrité absolue des racines postérieurs, des ganglions spinaux et de la mœlle épinière—*Arch. de Physiol.*, 1884, p. 231) has recently reported a case which was supposed to be locomotor ataxia, where after death no other lesion than neuritis of the peripheral nerves was found.

The disease known in Japan as *kak-ke* has been described by Scheube, who has carefully examined the nervous system in twenty patients. He found neuritis in every case; slight lesion of the cord, not sufficient to explain the symptoms, in only one case. He believes a specific poison is the cause of the disease.

Recently Dr. J. B. de Lacerda has published a monograph (see *Science*, March 14, 1884, p. 331. Etiologia e genesis de beri-beri. Pelo Dr. J. B. de Lacerda. Rio de Janeiro, 1884), in which he claims to have discovered the bacillus of beri-beri, and by injecting liquid containing this bacillus into rabbits and Guinea-pigs he produced a similar disease. He also found similar bacilli in rice, and cultivating he produced the organisms, and injecting these into animals produced the disease.

The nature of the pathological process in these cases was generally believed to be an inflammation of the nerves. But at a meeting of the Section for Psychiatry and Neurology of the Versammlung deutscher Naturforscher und Aerzte in Freiburg, held last Sept., Erb made two suggestions (*Allgemeine Zeitsch. f. Psych.*, etc., xl., 1884, p. 834), which he thought deserved more consideration:

"1. It is possible and probable that purely functional disturbances of the trophic centres in the spinal cord produce anatomical disturbances (degenerative atrophy) in the periphery (the motor nerves and muscles).

"2. It is possible that besides the complete destruction and separation of the trophic centres, yet other pathological processes occur in them which need not always go so far as to show themselves in the form of total degenerative atrophy of the peripheral regions of these centres."

These suppositions seem to have been answered sufficiently at that meeting by Strümpell, Schultze, and Seeligmüller, who show that Erb's hypotheses are less likely to be correct than that which refers the lesion to a peripheral neuritis. Erb replies that his hypothesis is only met by an hypothesis.

In the case just reported the degeneration in the peripheral ends is simply the degeneration due to the separation of a nerve from its trophic centre, the Wallerian degeneration (fig. 4 shows the first segmentation, fig. 5 shows the extreme degree of change). As specimens from nearer the origin of the nerves, this degeneration was less marked, and a larger number of healthy nerve fibres appeared. In those taken from the brachial plexus the nerves show change only about the constrictions of Ranvier. The first change is not segmentation, but a disturbance of the medullary sheath at these points (figs. 2 and 3).

The same change was seen in a specimen from a Guineapig, where the nerve was in the first stage of inflammation. Gombault (*Archives de Néurologie*, t. I., 1880, pp. 11–177) has studied the changes in neuritis produced artificially in Guinea-pigs by mixing lead with their food. He finds that the seat of commencing change in the segments of a nerve fibre is at the end of the segment, the axis-cylinder remaining intact. He concludes that the difference between inflammation and Wallerian degeneration is: (1) that in the same nerve fibre the lesion is discontinuous, it attacks certain inter-annular segments to the exclusion of the others; (2) the axis-cylinder is not interrupted, the myeline sheath and protoplasm are alone affected. It is a true parenchymatous inflammation; it may terminate in restoration of the fibre, or the axis-cylinder may be at length destroyed, and then the Wallerian degeneration follows in the distal end of the nerve fibre.

Gombault thinks this peri-axillary neuritis does not have its origin in a change of trophic action of the spinal centres.

Considering the changes found about the annular constrictions, it is reasonable to consider the disease a neuritis, not dependent upon changes in the spinal cord. The fact that the distal extremities of the nerves were most affected, and the change diminished toward the spinal centres and ceased in some nerves long before the spinal cord was reached, would also lead to the conclusion that the disease was not dependent upon changes in the cord.

In most cases the patients recovered more or less completely, but recovery was very slow in severe cases. It required much time and patience to straighten the contracted limbs, and strength was regained very gradually.

I am not sure that any treatment shortened the attack. Salicylic acid seemed to cut short the pain in some cases, and when it returned it was not quite so severe. It was necessary to give morphia to relieve pain. A 4 % to 5 %

solution of carbolic acid applied on cloths to the painful limbs was of decided advantage. One patient thought ice to the spine grateful, another preferred hot water, complaining that ice increased the pain in the limbs. Blisters over nerves seemed to be of advantage in one or two cases where used, especially in a case where the hyperæsthesia was very persistent.

It is well to try to avoid or diminish the extent of the contractions by position or gentle extension of the limbs, a sand-bag to rest the feet against, etc., for the contractions are difficult to overcome. To do so requires a persevering use of massage, extension, and electricity. These agents are to be used a long time to promote a return of power.

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CARCINOMA OF THE PERITONEUM.

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MOST malignant new-formations of the peritoneum are carcinomatous. Those of other nature are usually secondary, either by continuity or metastasis. Enchondroma has been thus observed, exhibiting a feature of malignancy unusual, though not without precedent (Wood, *Lancet*, 1881, i., 249). Myxo-sarcoma and sarcoma have also been encountered, both as primary and as secondary growths. Primary peritoneal sarcoma has been reported by Kelsch and Wannenbroncq,[1] Berlioz,[2] Weiss,[3] and others.

Carcinoma of the peritoneum may be primary or secondary. Of late years there has been not a little confusion concerning primary carcinoma of the peritoneum. From time to time peritoneal carcinoma has been observed where every evidence of its primary development was present at the necropsy. At the same time the impossibility of such occurrence continued to be urged by the upholders of the germinal lamina theory of tumor formation, by whom it has been insisted that carcinoma, a new-growth dependent for at least a portion of its structure upon tissues derived directly from the epiblastic layer of the blastoderm, could not be developed from the peritoneum, a purely mesoblastic structure.

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[1] *Prog. méd.*, ix., 1881, 729.

[2] *Jour. de la soc. de méd.*, etc., *de l'Isère*, 1879, 89.

[3] *Aertz. Berl. d. k. k. Allg. Krankenh, zu Prag.*, 1882, 128.

Various explanations of the apparent existence of primary cancer of this membrane have been offered. Some have assumed it to be really secondary to primary undetected formations in epithelial structures. Klebs suggested an infection of the peritoneum with epithelial elements, rather than by a carcinomatous new-growth originating in the peritoneal endothelium, leaving to future investigation the task of determining the route by which the epithelial elements are brought to the peritoneum.[1] These pathological difficulties have, fortunately, been greatly relieved by recent embryological discoveries, whereby practical experience may be reconciled with the prevailing theory. The investigations of Hertwig and Balfour show that the lining membrane of the peritoneum is derived from the hypoblast, and that it is, therefore, a true epithelial structure.[2] There is, therefore, no difficulty in concluding that carcinoma not only may but does occur as a primary peritoneal new-formation.

Varieties of Peritoneal Carcinoma.—The cellular elements predominate in proportion to the rapidity of growth of peritoneal cancer. In the most rapidly fatal varieties the growth may have the softness of brain substance. Acute miliary carcinoma is probably always medullary. In less acute primary carcinoma both this and the fibrous (scirrhous) and colloid forms occur. The connective-tissue element will be most abundant in the slower forms and hard leathery bands of malignant infiltration will be developed. Colloid cancer, usually indicative of a more chronic course, may complicate both the medullary and scirrhous forms. Petrina [3] records forty cases of peritoneal carcinoma. Of these fourteen were primary, nine medullary, and five scirrhous (fibrous); twenty-six were secondary—fourteen medullary, ten scirrhous, two colloid. Chuquet [4] found that of the cases collected by him, two thirds were primary. (He also found colloid carcinoma to be the most frequent. It is more correct to speak of this as a degenerative condition of cancer than as a distinct variety, and to apply the term colloid cancer only when the colloid change is predominating. To a limited extent it can be discovered in a large proportion of carcinomas.) Carcinoma may affect the peritoneum secondarily by continuity from other organs or tissues, and by metastasis. Melanotic cancer may thus rarely develop.

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[1] *Handb. d. path. Anat.*, 2d part, 1869, p. 337.

[2] Ziegler's "Path. Anat.," part 2. Translated by McAllister. Lond., p. 123.

[3] *Viertelj. f. d. prak. Heilk.* Prag., 1872, 2 B., 541.

[4] "Thèse de Paris," No, 548, 1879.

Symptoms and Course.—Primary peritoneal cancer may develop very insidiously, and has an indefinite and irregular course. Pain commonly first attracts the attention of the patient. This may become nearly constant or paroxysmal, most intense in some fixed region, and radiating thence to other parts of the abdominal cavity, or to the chest, shoulders, or back, or down the thighs. It is variously described as stabbing, stinging, burning, or as a dull, heavy sensation. As a rule it gradually increases in severity until it becomes a source of unending distress or even agony. It may, however, not be present during the earlier stages, or may never acquire especial prominence. This pain is, at first, not aggravated by pressure, and it is probable that the tenderness so often observed later may usually be attributed to peritonitis. About the period of development of pain, or sometimes even earlier than this, the patient realizes that he is not well. Vague disturbances of his digestive organs, anorexia, even disgust for food, and other signs of gastric indigestion, will appear with eructations, flatulence, etc. Vomiting may occur at this time, but more commonly when the disease is advanced, and is then often a result of peritonitis. At other times it follows primary cardiac or pyloric gastric cancer, of which the peritoneal growths are secondary results. Constipation will gradually become persistent and the patient will lose strength and flesh. At this time there will be no fever except in the more acute cases, and the thoracic organs will be unaffected, unless involved in a general carcinosis or subject to independent disease. In acute miliary carcinosis fever may be present from the first and the case closely resemble one of acute miliary tuberculosis.

Sooner or later the belly will become enlarged from the growth of the tumors, from meteorism, from ascites, or a combination of these conditions. In a large number of cases the tumors may be obscurely felt as nodules, varying in size from that of a nut to that of a child's head, deeply in the abdominal wall or more profoundly situated. At times the nodules are replaced by tracts of resistant matter not clearly definable, or by the hardened cancerous omentum crossing the belly as a broad band of induration. When felt, the tumors will usually be hard and resistant, except in the case of colloid cancer, when an obscure sensation of fluctuation may be perceived. Examination by the vagina and rectum will often assist the observer by revealing the infiltrations in the pelvic cavity. Not unfrequently the uterus will be immovably fixed in a mass of such material. The new-formations will often grow rapidly. It is uncommon, however, for them to be easily and definitely recognizable unless of large size, since they are obscured by ascitic fluid and by gas within the intestines. The occurrence of peritonitis will also tend to make a diagnosis difficult by forming adhesions whereby pockets of fluid and knuckles of intestines become fixed in various positions. The surface of the belly will thus be made uneven by the irregular distribution of cancer masses, localized meteorism, and encysted fluid. In the more acute forms it is not at all uncommon for the carcinomatous nodules to entirely escape recognition on account of their small size and wide distribution and the accumulation of ascitic fluid, and the true nature of the disease may not be determined *intra vitam.*

Chuquet claims that a symptom of highest diagnostic importance is the presence throughout the entire subcutaneous system and in the muscles, of " cancer granules " first described by Millard, and which are said to be perceptible to the touch. These "granules," however, do not seem to have been met with by other observers, and can therefore hardly be counted upon with confidence. What seems to be a somewhat similar condition has been described by Chvostek as a scattered crepitation over the belly in peritoneal cancer, or even in peritonitis, quite like skin emphysema. He attributed the sign to fluid enclosed in very small spaces with delicate and sharply limited walls, and which is forced out by pressure. He found it only where the abdominal viscera were adherent to the anterior belly-wall by peritonitic products containing fluid in very small cavities.[1]

Ascites is constantly present in these cases. It is due to peritonitis or to pressure exerted upon venous trunks within the belly cavity, or to hydræmia. It may vary in amount from one to twenty or thirty pints. The fluid is usually clear and of high specific gravity, with floating shreds of fibrin. Its character will often be ascertained through paracentesis performed for the relief of pain or for purposes of diagnosis. It will then be found, very often, to be tinged with blood or decidedly sanguinolent. This condition of the ascitic fluid is of the highest diagnostic importance, and has been insisted upon by a number of writers as indicating a strong probability of a cancerous origin. It must be admitted, however, that it is *possible* for this fluid to be sanguinolent in tubercular and even in chronic peritonitis. All things considered, sanguinolent ascitic fluid gives a strong presumption in favor of peritoneal cancer; and if the sedimentary deposit of this fluid be microscopically examined (a procedure first recommended by Foulis, of Edinburgh), the detection of groups of ordinary epithelial cells will serve to determine its cancerous origin.[1] The amount of fluid is not always proportionate to the duration of the affection. It is often irregularly distributed, by reason of the frequent peritoneal adhesions, which also bind the intestines in such a manner that the usual position of these in simple ascites is not often observed. Another rare peculiarity of the ascitic fluid of carcinoma has been observed by Quinecke, Klebs, and Brieger. Here the fluid is milky white, and forms a creamy surface layer of fat-corpuscles and granular fat. Such cases have been supposed to represent chylous ascites. Brieger, however, thinks that there is no escape of lymph, but that the appearances are due to fatty degeneration of peritoneal epithelium. After paracentesis, peritoneal friction sounds may sometimes be heard. General dropsy frequently occurs later, though œdema of the lower extremities has been known to precede ascites. Pericardial and pleural effusions may arise from cancerous metastasis or extension, and the abdominal wall may become œdematous. Œdema of the lower extremities from pressure upon large intra-abdominal veins may become very intense, and may lead to erythematous, erysipelatous, or even gangrenous inflammation. When pressure is exerted upon the ascending vena cava, the veins of the abdominal wall become very large and tortuous, with reversed blood-current.

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[1] *Œsterreich. Zeitsch. f. prakt. Heilk.*, 39, 1866.

It has been said that constipation is nearly always present early in the disease. This will be associated with more or less localized meteorism. These accumulations of gas sometimes press the diaphragm above its natural limits, and add greatly to the general discomfort. Attacks of diarrhœa may alternate with constipation, and toward the end diarrhœa may become persistent. On the other hand, the lumen of the bowel may ultimately be destroyed, and the patient perish with the symptoms of obstruction. Hepatic and urinary disorders, as direct results of the carcinoma, are not constant. Jaundice may be occasioned by pressure of the new-growths upon the bile-ducts. The renal functions are not often disturbed. The urine will often be highly acid and deposit urates freely. Micturition may sometimes be painful and frequent from implication of the bladder.

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[1] Beatson, *Glasgow Med. Jour.*, 12, 1879; Chuquet, *loc. cit.;* Brieger, *Charité-Annalen* viii, 1883, 109.

While these symptoms are developing, the patient's general condition becomes markedly worse. In a short time, possibly several weeks, more often several months, the various disturbances of nutrition, together with the rapid increase in number and size of the cancerous growths, will have induced that peculiar condition known as the cancerous cachexia, which will, of itself, often direct attention to the true nature of the disease in obscure cases. Tenderness to pressure will be superadded to the ever-increasing distress. Rapid emaciation and increasing debility result from diminished assimilation, pain, sleeplessnes, and general discomfort. The inguinal glands may become enlarged and indurated, and metastatic deposits may occur in other parts of the body. Cancerous infection of the tract of puncture of paracentesis has been observed by Brieger, Reinke, Quincke, Unverricht, and Chuquet. Fever, which may have been present from a very early period, will sooner or later appear and become constant. Pain, insomnia, nausea and vomiting, alternating constipation and diarrhœa, profound debility combine to intensify the sufferings of the patient until death occurs, from the development of the cachexia, from pulmonary œdema, pneumonia, perforation of the bowels, peritonitis, hemorrhage, or some intercurrent affection.

When the peritoneal carcinoma originates through continuity or metastasis, its symptoms will blend with those of the primary affection, and will often play a *rôle* quite unimportant ; or it may become diffused with great rapidity. In such cases, of course, the symptoms of the primary disorder will not be mitigated by the involvement of the peritoneum, but may be less sharply defined than when uncomplicated with those of secondary formations.

Duration and Prognosis.—Peritoneal carcinoma probably runs its course more rapidly than any other form of cancer, ending fatally in from four to six months. Rarely cases terminate within a few weeks after the *apparent* beginning of the malady. On the other hand, life may be prolonged for a year or eighteen months. Vidal reported a case that lasted two and a half years (Chuquet). This seems to be the extreme. Petrina gives the medium duration of primary peritoneal carcinoma, reckoning from the earliest fever, as six weeks, and of secondary cancer as from one to three months. The shortest course observed by him in primary carcinoma was one week ; the longest, six months ; and in secondary cancer the shortest duration was three weeks ; the longest, eighteen months. Other authorities give a longer average duration to the disease, though all agree that the fatal termination will generally come within six months.

Etiology.—This is very obscure. Traumatism has been ascribed as an occasional cause of primary carcinoma of the peritoneum, but this is very doubtful. Probably, also, chronic peritonitis rather occurs as a consequence than as a cause of the new-formation. Heredity exerts a positive but indeterminate influence. Although there seems to be no time of life at which peritoneal cancer may not occur, it is prone to appear after middle life. It has been observed in infancy and even in the fœtus. The following tables of

Petrina and Chuquet show the ages of the patients recorded by them.

	PETRINA.		CHUQUET.	
Age.	*Male.*	*Female.*	*Cases.*	*Total.*
10–20	—	1	1	2
20–30	1	2	9	12
30–40	—	2	5	7
40–50	6	2	4	12
50–60	4	7	13	24
60–70	3	7	4	14
70–80	2	3	5	10
	16	24	41	81

It thus appears that nearly one third of all cases occurred between the ages of fifty and sixty, and three fourths of all after the fortieth year. Sex has a decided influence, females having a much greater liability. Of Petrina's forty cases, sixteen were males and twenty-four females. Chuquet records the sex of forty-four cases, of whom seventeen were males and twenty-seven females. The greater liability of females is probably due to the comparative frequency of cancer of the female genital organs, and its extension to the peritoneum. Secondary cancer arises by extension or metastasis from other parts or organs. Chuquet concludes that the centres most often the seat of the primary deposits, are, in order of frequency, ovaries, stomach, liver, and uterus. Petrina's tables show, however, the stomach and pylorus to be the most frequent primary centres of twenty-nine cases of secondary peritoneal cancer, the stomach and pylorus were primarily affected in seventeen, the ovaries, Fallopian tubes, and uterus in four, the liver in three, the pancreas in two, the mesenteric and retroperitoneal glands in two.

Diagnosis.—This is frequently of extreme difficulty, especially in primary carcimoma ; indeed in rapidly fatal cases the nature of the disease may be only determinable after death. At best, the earlier stages usually escape recognition. The affections most apt to be confounded with peritoneal cancer are peritoneal tuberculosis, cancerous and other tumors of the abdominal viscera, ascites from cirrhosis, hydatid and ovarian cysts, meteorism and impacted fæces. When the malady is secondary it is not usually difficult to attribute the new symptoms to their true cause. Their appearance, more or less complete, in a person known to have gastric, ovarian, hepatic, or other variety of cancer, will readily be recognized as indicating an extension or metastasis. Cancer secondary to undetected primary growths will not differ from primary forms in the difficulties of its recognition. Tubercular peritonitis may present the closest clinical analogies with rapid peritoneal carcinoma. There may be the same early development of ascites, the rapid wasting and debility, the abdominal pain, even the formation of tubercular masses detectable through the abdominal wall. Diffusion of nodules to other serous membranes may occur in both affections. Carcinoma, however, may be, indeed, is often quite devoid of fever, which, when present, is quite irregular and unlike the fever of tuberculosis, the evening exacerbations of which are characteristic. The ascites of cancer is more abundant than that of tuberculosis, though in both affections the fluid may be irregularly encysted through inflammatory adhesions. Although pain is present in tuberculosis, it is altogether less than in cancer where it seems often quite out of proportion to the amount of appreciable disease. The existence of tubercle in other organs and the presence of persistent diarrhœa, should give a prejudice in favor of similar disease of the peritoneum. This, however, should not be held too rigidly. Cancerous tumors of the stomach and liver are usually unaccompanied by ascites, unless the peritoneum becomes involved, or secondary deposits occur, compressing the vena portæ or ascending vena cava. In the latter case œdema of the lower extremities will also be present. Hepatic cancer will also show the influence of the diaphragm in following the respiratory movements. Sarcomatous tumors of the peritoneum or intra-abdominal organs, or of the retro-peritoneal glands, do not afford well marked clinical differences with carcinoma. Simple tumors are to be distinguished by their benign course. Ascites from hepatic cirrhosis is of more chronic course, is more painless, and presents altogether a different clinical history.

The apparent cachexia of cirrhotic patients may at times be misleading; but the symptoms of hepatic contraction will be present, and the absence of tumors may be ascertained through paracentesis. Hydatid and ovarian cysts have a slower development and a localized development of fluctuation. They are, also, usually painless, unless complicated with peritonitis. The hydatid thrill, when present, will assist the diagnosis. It should not be forgotten that ovarian cysts are sometimes associated with carcinoma.

Meteorism may distend the belly-wall, but the pure tympanites, and its dispersion under appropriate treatment, will distinguish it. Also, fæcal accumulations may be recognized by their plasticity, and their removal by proper purgation.

The presence of painful intumescence of the belly (with or without fever), with irregularity of surface caused by associated areas of dulness and resonance from fluid, gas, and tumor formations of rapid growth, with emaciation, pain, and the development of cachexia, will usually suffice to distinguish peritoneal carcinoma. Ascites may, however, be so intense as to prevent the recognition of other physical conditions, in which case paracentesis may be resorted to. This should be done with due caution, as it is sometimes followed by unhappy results, especially in cases of colloid cancer (Faucon : *Jour. des sci. méd. de Lille*, iii., 177). The operation is contra-indicated where the physical conditions allow a tolerably certain diagnosis to be made, and should never be done unless the patient be confined to the bed and vigilantly cared for for several days afterward. If, after paracentesis, a solid tumor be detected, the presumption in favor of its malignant nature is strong. The character of the ascitic fluid is of the utmost importance. It may be like ordinary ascitic fluid, but it is very often sanguinolent, a condition almost characteristic of malignant disease. The detection of epithelial cells, such as occur in carcinomatous growths, in the sediment of this fluid, may be considered as conclusive of its cancerous origin.

Pathological Anatomy.—In necropsies, after the more acute primary cancers of the peritoneum, the nodules may be found scattered over every part of the membrane. They may be no larger than millet-seed, and of a whitish or grayish color, and without inflammatory areola. Miliary tuberculosis may be closely simulated. Usually, the nodules vary within much wider limits, often attaining the size of hen's eggs or oranges. They remain sharply circumscribed, or merge into masses of greater or less extent, sometimes forming great plates of infiltration. In the mesentery, Chuquet asserts that the nodules commonly are situated near the points of intestinal attachment. Nodules of the size of a grain of wheat, or of a pea or bean, are very apt to have central depressions, differing thus from tubercle. In the process of growth they tend to assume a spherical form, and may even become pedunculated, showing a resemblance to clusters of white currants (Bristowe); or the pedicles may become reduced to mere threads, and indeed, as in a case reported by Matthews Duncan (*Med. T. and G.*, 1872, ii., 432), they may be severed and the nodules float free in the abdominal cavity. Sometimes the new-growth at first looks as though drops of melted white wax had fallen upon the membrane and there hardened, an appearance also met with in cancer of the intestinal mucous membrane. When diffused infiltration of the membrane occurs it may, in proportion to the rapidity of its development, have the characters of medullary or of scirrhous cancer. The colloid change may affect either variety.

In medullary cancer, soft, brain-like masses may line the parietal peritoneum forming infiltrations of great thickness; or, involving the peritoneal coat of viscera, may deeply imbed them, sometimes without involving their structure. The greater omentum may thus become greatly thickened. Such masses may form, with inflammatory products, conglomerations of bowels, peritoneum, and viscera. This is especially the case in the pelvic cavity. The new-growths will be of light or dark gray or reddish color, with often brownish centres from extravasation. Scattered throughout the masses little cystic accumulations of clear gummy fluid may often be discovered. A granular or mammillated aspect will be presented, while in structure such friability may be presented that the infiltrations readily break down under the finger. Masses may attain the appearance and size of the adult human brain.

In more chronic primary carcinoma, the new-growth may be more localized and the tumors be much fewer in number, though sometimes reaching great size and weighing several pounds. The softer varieties are usually very vascular. In the scirrhous forms, the peritoneum may be thickened by a dense infiltration, giving it a leathery appearance; and the great omentum, reduced to a contracted and thickened band, will stretch across the abdominal cavity. Colloid cancer, which will be frequently met, will be recognized by its semi-fluid, gelatinous appearance.

It is apt to form great masses, and to be widely diffused. In all varieties the peritoneum will often be opaque and thickened almost throughout. Primary peritoneal cancer may also be metastatic, attacking neighboring parts or viscera; or, it may spread by continuity, growing into adjacent organs from the surface, or, extending through the diaphragm, involve the pleuræ. Sometimes organs are completely destroyed by the infiltrations. This is especially the case with the ovaries.

Secondary cancer of the peritoneum will usually be found most densely distributed near the primary centre, whence it has extended by continuity. In such cases, the colloid form is very frequently observed. The anatomical conditions will only differ from those of the primary forms in the superadded pathological changes. The intestines will show various degrees of alteration. They will be more or less constricted, the narrowing reaching its highest grades in the cases where the mucous membrane was primarily affected. Swelling and œdema of the mucous membrane are often found. In all cases evidences of peritonitis will rarely be absent; and masses will sometimes exist where central softening has occurred. Perforation of the bowels, with escape of their contents into cavities thus formed, is not unknown; and limited areas of fatty degeneration and calcification occasionally are detected. Evidences of hemorrhages, of greater or less extent, are not unfrequent.

Treatment.—As the affection runs a necessarily fatal course, treatment must be directed toward the alleviation of suffering and the assistance of the powers of assimilation. Other indications for treatment must be met as they arrive. No treatment addressed to the carcinoma itself will avail.

A CASE OF ACUTE POLIOMYELITIS IN THE ADULT.

By JOHN VAN DUYN, M.D.,

SYRACUSE, N. Y.

History: F. D., a clothier's clerk, twenty-three years old, of average height and good muscular development. Had worked on a farm during summers until 1880, which year he spent at school. Early in 1881 he entered a clothing-store as clerk, but in the following November he had malarial fever, and in February. 1882, had a moderately severe attack of typhoid fever: From the time of his recovery, in early spring, until the following October, he worked on a farm, and then resumed his position as clerk. Had never had venereal disease, and had never been intemperate in the use of alcohol.

Aug. 24, 1883, he walked nine miles, arriving at his destination at two o'clock the next morning, went to bed at three o'clock, and slept six hours. At noon, he engaged with others in "putting" a stone, which weighed nearly forty pounds, and in jumping. The stone was "put" from the shoulder by the right arm forward, and thrown by both arms over the head backward. This contest was most violent and lasted two hours. Then he ate a hearty dinner. At 4.30 P.M. he walked over three miles, then rode in a stage two miles, and in the cars ten miles, after which he walked with friends in the city, and finally went to bed at eleven o'clock. The next day he felt some soreness, which, in a day or two, entirely passed away. Until the end of the two weeks following, his life was as it had been before. By his exertion he did not become overheated, was not chilly, nor did he take cold. On September 7th, when he woke in the morning, he felt severe pain between the shoulders, equal in intensity on both sides of the spinal column, which was greatly increased by turning the head to either side. He went to work, but during the day he felt chilly and his face was very red. In the afternoon the right hand began to lose its grasp, so that he could hardly hold the pencil in making sale-tickets. The power to use this hand grew gradually less, until, three days later, it disappeared entirely. That evening he rode ten miles by rail. He could not sleep because of headache, chills, and nausea. The soreness became universal. The second day, he got up, could raise the right hand to his face, but could not button his clothes with it. He walked to see the village doctor, but found the strength of his legs failing to the extent that, in the afternoon, when he rode home, three miles distant, he could with difficulty get into, and out of the wagon. Went to bed at night and "took a sweat." The third day, felt sick and sore, but could get up and even went out of the house. When he tried to put on his hat with his left hand, the right being powerless, he found it impossible, and with difficulty only could he lift this hand to the latch of the door. After he had gone to bed at night he found he could not raise either elbow from the bed. From this time on he was confined to the bed. He suffered from general soreness. To breathe gave him pain, and he could not lie on either side, because of the pain which the pressure on the chest gave him. The whole body, excepting the head and neck, was so sensitive that suffering was caused by the gentlest touch. During the following three or four days paralysis of motion became general in the extremities, but not affecting the head, neck, thorax, and the left forearm and hand. Could not raise his heels from the bed, but while on his side, could partially flex the thighs on the abdomen. Constipation became severe, but the bladder was unaffected. During this time the temperature was twice observed and recorded. On the fourth day it rose to 102°, a few days later to 101°. After the tenth day improvement began to be manifest, and continued until the end of the second week, when he could get into a chair, but could not stand, nor could he raise his feet from the floor. The soreness had receded to the shoulders. At the end of the third week, could walk with assistance, but could not lift the feet higher than enough to prevent dragging. Constipation continued and did not disappear until two months later. The right arm was powerless. The left arm was recovering strength rapidly. This arm had not been affected below the elbow. At the end of the fourth week he could walk alone, the right leg having regained considerable strength, but the left leg could as yet be moved only with difficulty. At this time he began to feed himself again, by using the left hand after the elbow had been rested on the table. Throughout there had been no abnormal sensations, no numbness. During these four weeks he lost sixty pounds in weight.

Examination, Jan. 12, 1884: Complete loss of voluntary motion in, and great atrophy of all the muscles supplied by the right brachial plexus, excepting those supplied by the fifth and sixth cervical roots. The right hand, with extreme atrophy in all parts, was *en griffe*. The muscles of this extremity, including those of the shoulder, were unaffected by faradism, but all, with the exception of those in the hand, reacted to strong galvanism. On the left side the supra-spinatus and infra-spinatus were greatly atrophied, the pectoral muscles less so, while all contracted under faradism. The elbow could with greatest difficulty be raised to the level of the shoulder. Below the shoulder the left arm was normal. Power to raise either knee was diminished. The left anterior tibial was not under control of the will and was atrophied, but reacted to strong faradism. Because of the loss of this muscle the foot falls in extension when raised.

To the present time, May 16th, treatment by electricity has been almost daily. All the muscles of the right upper extremity contract under galvanism, with the formula CCC. > ACC., while the brachialis anticus, biceps, sup. longus, and pectorales major and minor contract under faradism. The pectoral muscles, biceps, and anterior brachial show decided contractions by the will. The nutrition of these muscles under the influence of the will so far exceeds that of the others that their growth causes them to stand out in marked contrast with the surrounding atrophy. The will has control of all the affected muscles of the left shoulder and of the left anterior tibial.

This typical case of acute spinal paralysis is recorded because of the clear history of the progress and retrogression of the paralytic state, and the manifest relationship between the disease and the violent muscular exertion which preceded it. Should the gymnastic violence be conceded as the cause, than which no other is apparent, then interest must attach itself to the length of time which intervened between the cause and the outbreak of the disease, and to the fact that while both extremities, upper and lower, were affected during the fever, the muscles which remained paralyzed and atrophied were those most violently exercised, and the severity of the results and their permanency are in direct ratio with the use of the muscles.

AN UNIQUE CASE OF POLIOMYELITIS ANTERIOR ACUTA OF THE ADULT.

By AMBROSE L. RANNEY, M.D.,

PROFESSOR OF APPLIED ANATOMY IN THE NEW YORK POST-GRADUATE MEDICAL SCHOOL AND HOSPITAL.

I DESIRE to call attention to a case which has lately come under my personal notice, and which seems to present some very striking peculiarities. I refer to a marked attack of poliomyelitis of the acute (?) form in the adult, which was followed by a paralysis of the leg and arm of opposite sides. Although in the literature of the subject different forms of paralysis—such as monoplegia, paraplegia, hemiplegia, and paralysis of all four limbs—have been recorded, I am unaware of a case where there has been the peculiar combination of symptoms which I now record. In Seguin's brochure upon this disease, published in 1874, he cites from Duchenne four cases, three of which had all of the limbs affected, while one developed paraplegia. Those selected also from Charcot and Gombault, as well as the personal cases cited by that author, do not contain a single instance of a similar type. In my search through the various text-books and the journals published since that date, I have failed, as yet, to detect a parallel instance. If I am correct in my belief and in my diagnosis, the case presented is unique.

The history of the case is published as it was noted at the time of my clinical examination. I hoped to be able to publish a more detailed statement in regard to the exact measurements of homologous parts, the electro-muscular reactions, etc., but I was disappointed by the patient failing to keep an engagement made with me for that object. I was unable also to obtain a photograph of his limbs for the same reason.

The history obtained from the patient is given in full.

A young man presented himself at one of my clinics in Burlington, Vt. (April 27, 1884) with the following statement of symptoms :

Age twenty years. Unmarried. Occupation—that of a laborer. In the summer of 1882, patient was employed in a saw-mill, and was obliged to run a treadle with his left foot and to push boards against a circular saw with his right hand. Shortly previous to the time of the development of his nervous trouble, he was severely stung by a swarm of bees upon the exposed portions of his body. He noticed no particular results from the accident, however, and the wounds healed kindly. After a day's fishing, the patient slept upon the wet grass and was attacked immediately with a marked febrile disturbance, which was followed by a paralysis of sudden development.[1] The paralysis was confined exclusively to the *left foot and leg*, and the *right upper limb* (the ones which he constantly employed in his daily vocation). The leg-paralysis disappeared with great rapidity, and in a few weeks he was able to use the right and left lower extremities with equal power. The right arm, however, never regained its power, and began to give evidence of rapid wasting of the muscles of the shoulder and hand. He has never noticed a wasting of the muscles of the lower extremity.

On examination, the grasping power of the right arm was almost abolished ; the muscles of the thumb were atrophied to such an extent as to allow the fingers of my hand to perceive each other with distinctness when they grasped the hand between the metacarpal bones of the thumb and the index-finger. The "bird-claw" deformity of the hand was partially developed, but the interossei showed a less-marked wasting than existed in the thenar eminence. The deltoid was almost completely destroyed, and the muscles of the scapula were also affected to a marked degree. The circumference of the limb was less than that of its fellow. A very strong faradic current produced slight contractions of some of the muscles of the hand and forearm, but the entire limb seemed to respond very feebly to the current, and no electro-reaction occurred in the muscles of the thumb. No constant-current battery was at hand; so that the "reaction of degeneration" was not positively demonstrated, although it would undoubtedly have been found in many of the muscles.

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[1] The patient stated at first that the paralysis came on "inside of a week" ; but on being questioned sharply, he stated that "he was not sure but that it occurred in two days." He was positive, however, that it developed not more than seven days after sleeping on the wet ground.

The lower limbs exhibited no atrophic changes or loss of muscular power. The sensibility to tactile sensations and the impressions of pain and temperature were normal in all of the limbs. A careful examination was made of these points. The patient had been subjected to massage and the faradic current at intervals for months, but without apparent benefit. The bladder and rectum had never given any evidences of impairment.

Certain peculiarities in this case struck me, as follows:

1st. The continued use of the right hand and left leg seemed to act, to some extent, as an etiological factor in the case. The cord was apparently attacked in those segments that had been subjected to excessive and continuous use, and the lateral half of the cord had been involved in each instance. The left arm and right leg, which were not employed in his occupation, were not attacked.

2d. The leg-paralysis recovered quickly and perfectly; but the arm and hand never regained their power. He had been severely stung upon the arm but not upon the leg, although this may not have been more than a coincidence.

3d. The cells of the anterior horns in the cervical segment degenerated rapidly, as evidenced by the rapid atrophy of the shoulder and hand, while those of the lumbar segment experienced no permanent impairment and regained their function in a few weeks.

I can see no elements of doubt as regards the diagnosis of this case. The exposure, the febrile symptoms, the paralysis of sudden advent, the subsequent atrophy of muscles, and the monoplegic type of the palsy, coupled with the fact that the limbs involved were on opposite sides of the body, can be explained to my mind in no other way. The quickly-developed loss of farado-muscular reaction also adds a crowning feature to the list of symptoms. The case seems to be a particularly unique one of this comparatively rare form of disease.

ANALYSIS OF SEVENTY-FIVE CONSECUTIVE CASES OF POSTERIOR SPINAL SCLEROSIS, WITH SPECIAL REFERENCE TO A SYPHILITIC ORIGIN.

By E. C. SEGUIN, M.D.

IN the last three years a considerable number of observers, European and American, have recorded the statistics of cases of posterior spinal sclerosis, with especial reference to its relations to syphilis. And every addition to these statistics, even if limited in numbers, is help toward the elucidation of the question of the etiology of tabes. Through extensive coöperative work of this kind, we may reap scientific and even practical results of some value. This consideration has led me to go through my case-books and contribute my mite to the subject.

The cases of tabes which have come under my observation since 1866 are naturally divisible into two classes: *i. e.*, those seen in private practice, and those observed at my clinic for diseases of the nervous system at the College of Physicians and Surgeons. The last named cases were examined and published by my friend, Dr. Birdsall, last year,[1] and I do not care now to reproduce that tabular statement. Suffice it to say, that Dr. Fuller, one of my clinical assistants, went to the trouble of going over the clinic case-books again, and reached the same numerical results as Dr. Birdsall.

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[1] "Proceedings of the American Neurological Association," meeting of 1883, *Journal of Nervous and Mental Diseases*, July, 1883.

I now propose to present the results of an analysis of the cases of tabes which have been seen by me in private practice.[1]

The total number of cases was 75, including a number in the first stage, and a few which, while exhibiting many of the symptoms of tabes, were not typical.

I am surprised to find that in a large proportion no mention was made of syphilis.

No mention made of syphilis,	21 cases	28 %
Syphilis mentioned	54 "	72 %

It is therefore better to exhibit the further proportionate results in a double aspect.

I. With reference to the total number of cases of tabes recorded:

Syphilis not mentioned in	21 cases	28 %
No chancre or syphilitic symptoms in	15 "	20 %
History of chancre or syphilis in	39 "	52 %
	75 "	100 %

Thus in more than one half of the total number of cases a distinct history of syphilis or of chancre was obtained.

II. In the more correct relation to the number of cases of tabes in which a positive statement was recorded on the point in question:

No syphilis or chancre in	15 cases	27.78 %
History of chancre or syphilis in	39 "	72.22 %
	54 "	100.00 %

This very large percentage is somewhat in accord with the results of Erb, Fournier, Voigt, Rumpf, and others.

For those who still believe in the distinct duality of the syphilitic poison (*i. e.*, in the existence of non-infecting chancres), the following additional summary may not be without interest:

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[1] These statistical results were presented by Dr. Birdsall, on my behalf, to the Am. Neurological Association at its recent meeting in June.

Cases in which chancre alone occurred	23	42.59 %
Cases in which chancre and secondary syphilis occurred,	16	29.63 %
Cases in which there was no history of syphilis.	15	27.18 %
	54	100.00 %

At first sight this exhibit might seem to militate strongly against the syphilitic origin of tabes; but upon further consideration, when one reflects upon the very large proportion of cases of common (*i. e.*, non-systematic) cerebral and spinal lesions, in which we only obtain a history of chancre, followed by years of good health, then by the nervous lesion, it becomes necessary to drop this objection. But even admitting that there are harmless "soft" chancres, we also know that in a large proportion of cases of "hard" chancres, skilful treatment may indefinitely postpone so-called secondary symptoms.

EDITORIAL DEPARTMENT.

REMOVAL OF THE UTERINE APPENDAGES.

A LETTER

By LAWSON TAIT.

I have written so much on this subject, that I am almost ashamed of putting pen to paper about it again ; but I am so constantly subjected to misrepresentation, that it is difficult to resist the inclination to reply. The article[1] by Dr. William M. Thallon obliges me to ask for some space, for whilst he gracefully credits me with far more than I deserve, he also to some extent misunderstands what I have written, or he has taken my views second-hand.

In the first place, I object altogether to the title of his paper : "The Battey–Tait Operation." The method of applying names of persons to particular proceedings is a constant source of error, and it clearly is so in this instance. I have no objection to be bracketed with my distinguished friend Dr. Battey, but as the principles of our proceedings differ entirely, such a union as is proposed by Dr. Thallon is wholly inconsistent. Dr. Battey proposed the removal of the ovaries for the purpose of bringing on artificially the menopause, for the purpose of influencing certain indefinite reflex symptoms. This proposal I have followed five times, and with such bad results that I have said over and over again in published papers that I should not continue the practice. Dr. Thallon is not justified, therefore, in saying that I have done hundreds of such operations, that Spencer Wells has done only one or two, and Keith one or two. Dr. Thallon is mixing up things that are wholly distinct, and I have protested against this mixture very many times.

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[1] Vide editorial in April number of ARCHIVES.

The principle of the operation I advocate is to save life and relieve suffering by the removal of diseased organs, or for such conditions of physical disease as appear, on theoretical grounds, sustained by actual experience, to justify the proceeding. I rarely operate save in the presence of physical signs ; and in the exceptional instances of operations performed for subjective symptoms only, I carefully guard myself by elaborate consultations.

Let me first speak briefly of Dr. Thallon's conclusions, and then I may say a few words on some passages in the text of his paper.

His first conclusion is that "the operation (Battey–Tait) is not justifiable for purely nervous symptoms, independently of clearly-marked evidences of organic ovarian disease." I wholly agree with this, only I say this is a criticism of Battey's operation, and not Tait's.

Dr. Thallon's second conclusion is that "it is not justifiable in cases of uterine disease or hydro-salpinx."

In answer to this I say that my proposal to remove the appendages for uterine myxoma, made almost simultaneously by Hegar and myself, and therefore, on Dr. Thallon's plan, entitled to the name of the Hegar–Tait operation, has been in my hands a brilliant success. Mr. Knowsley Thornton has found it so successful that he says it should always be a preliminary to hysterectomy. I agree with him so far, that I believe if it were so, there would be a very small number of cases left for hysterectomy. The success of this proceeding has been so great, and has received such abundant confirmation, that I cannot imagine how Dr. Thallon gets his conclusion.

As to hydro-salpinx, I have written so much on this point that I can only feel that Dr. Thallon has omitted to consult my writings. As it is wholly impossible to diagnose with accuracy the existence of hydro-salpinx without abdominal section, as it is wholly impossible to be sure of its position, tapping is altogether out of the question. The recommendation of tapping as an alternative for removal of the diseased tube, can come only from one who has no practical acquaintance with the disease.

On this point let me just interpolate a complaint against one sentence of Dr. Thallon's article, in which he says : " Mr. Lawson Tait in especial has been singularly rash in minimizing the risks, and he has probably done a great deal of harm in encouraging men to undertake a so-called 'easy operation,' which too often has ended fatally for the patient."

I protest against this as most unfair. I have never said it was an easy operation. I have always said it is one of the most difficult things in surgery. I have never encouraged any one to do these cases unless he was prepared to do as I have done—give up every thing else to follow them. I have always said, and I say again, that a man whose prospect is to do only five or six cases of this kind in a year has no right to attempt them, and that if he does and they are fatal to the patients, he has done a very wrong thing. I have never minimized the danger, I have constantly emphasized it. I have never said that my own mortality, about one per cent., could be reached by any body save by doing as I do, devoting my life to the work, and operating five, six, or eight times a week.

Dr. Thallon's third conclusion, that the operation of removal of the uterine appendages for the relief of uterine hemorrhage, or the arrest of uterine tumors, I have already answered.

Dr. Thallon's fourth conclusion I accept with more than pleasure, as it is conveyed in words extremely gratifying to me. I have had a hard fight during eight years to establish my position, and now, thanks to Dr. Kingston Fowler, of the Middlesex Hospital, it has been completely established. That my work is accepted as " one of the most glorious achievements of the surgery of our age " is reward enough for all my trouble.

Dr. Thallon raises the much-discussed question as to the influence of removal of the ovaries and tubes in menstruation. Let me say here again what I have recently said completely, and several times incompletely. Removal of the ovaries alone has little or no influence in arresting menstruation. Removal of the tubes alone arrests menstruation in five out of six cases. Removal of both tubes and ovaries has the same result as removal of the tubes. There are some cases in which even removal of the fundus uteri together with both tubes and ovaries exerts no influence whatever on the periodic regularity of the menstrual flow. In the course of a few days a paper will appear in the *Medical Times and Gazette* comprising the doctrine of Ritchie and Reeves Jackson, that menstruation and ovulation are wholly independent functions. Dr. Thallon seems never to have come across even a suggestion of this before, and, therefore, much of his criticism of my own views I cannot answer here for want of time and space. But if gynæcologists for the future will approach this question with the glimmering of an idea that it is just possible the two functions may not be dependent one on the other, much trouble, confusion, and repetition will be saved.

As to the influences of the removal of the uterine appendages for masturbation or nymphomania, I am quite satisfied that the prospect of good being done is very small. I have, therefore, persistently refused to operate in such cases, as I have not found that removal of the appendages has deprived women of their sexual appetite. Only a few weeks ago, at the Harveian Society, Mr. Bryant of Guy's and myself were able to quote cases where supravaginal hysterectony did not in the least interfere with the sexual appetite. Even if removal of the uterine appendages did destroy it, I for one would not hesitate to operate for the cure of disease ; and I would look upon any man as an inhuman brute who would refuse relief to his suffering wife because his lusts would have some smaller gratification. The only men who would take such a view of their position would be those who had brought the troubles of hydro-salpinx and pyo-salpinx on their wives as the result of gonorrhœa. To me it becomes more and more amazing that this disgusting argument should be discussed. It is our duty to save life and prevent suffering, not to pander to the degraded desire of the debauché.

Dr. Thallon points out what is perfectly true, that, whereas, I do a hundred such operations, Sir Spencer Wells does one or two, and he further says that we must see about the same class of cases. I admit this, but I plead that I am a generation younger than Sir Spencer Wells, and I admit that when my time comes I shall probably have the same difficulty in taking up a new theory that Sir Spencer has shown in the present case. Some months ago Dr. Horrocks asked me at the Obstetrical Society how it was I saw so many cases of pyosalpinx and that they saw none at Guy's Hospital. My reply was that there must be large numbers of them at Guy's Hospital but that they were not discovered. Dr. Thallon says that I take particular delight in saying that in many of my cases operation had been previously refused in London, and if Dr. Thallon knew how much I have been misrepresented and maligned by some of my London brethren he would not be surprised. But my delight has been greatly intensified by a paper read last week by Dr. Kingston Fowler, which proves up to the hilt every thing I have said on the subject, and completely covers the ground raised by Dr. Horrocks, and justifies my answer to it. In the discussion on Dr. Fowler's paper, Dr. W. S. Playfair said that a middle course between the practice at Birmingham and that of those who denounced the operation was the safe one. He illustrated this safe course by his own proceedings, which resulted in the awful mortality of twenty-five per cent. All I can say is that I hope I may never fall into this safe course of Dr. Playfair, for if I do I shall speedily give up practice. No man is justified in operating in such a way. What I constantly plead is that these operations can never be justified save by a very low mortality. If my mortality went up to twelve per cent. I should stop.

I entirely endorse what Dr. Thallon says about the cases where the operation is done for "pain, epilepsy, or insanity." It is too much of the nature of a leap in the dark to have my support. I do not condemn it, however, for there have been some brilliant successes, and there ought to be no mortality at all when there is merely a removal of normal appendages. Speaking of a case related by Dr. Fordyce Barker, Dr. Thallon says he thinks I would not have hesitated to operate in it. He is entirely wrong, for as he gives the case, I should certainly have refused, and have done so in several very like it.

Upon two points let me say that I entirely agree with Dr. Thallon. He says: "Where, however, one ovary lies so low down as to be the first thing encountered by the finger after entering the vagina, especially if it is bound down by adhesions, or by a heavy and retroverted uterus, the chances of any thing short of the radical operation effecting a cure are very slight." This is absolutely true, and some of my most brilliant successes have been in precisely such cases.

Again, Dr. Thallon says: "I believe it is, broadly speaking, true that the more extensively the ovaries are matted up in the products of an old peritonitis, the greater will be the necessity for the operation." This is quite correct, but I cannot at all agree with Dr. Thallon when he further states that this "suggests the unpleasant reflection that the greater danger is there of it being fatal."

In conclusion I have only to say that I have examined with great care the contents of many Fallopian cysts and have never seen, though I have specially looked for, columnar ciliated epithelium. The suggestion of Dr. Frank Ferguson has therefore been anticipated and exhausted.

BIRMINGHAM, *May* 6, 1884.

DR. THALLON'S REPLY TO MR. TAIT.

The answer of Mr. Lawson Tait to some points in my article is chiefly interesting in what it admits. I am extremely glad to find that two of my four conclusions are so heartily endorsed by him. I think it will certainly surprise some of the enthusiastic gynæcologists who have been doing "Tait's operation" for all sorts of purely nervous symptoms ("pain, epilepsy, and insanity") to learn that Mr. Tait himself "wholly agrees" with me that the operation is not justifiable for this class of cases,—and that "it is too much of the nature of a leap in the dark to have my (*i. e.* Lawson Tait's) support."

That declaration is decidedly more explicit than any limitation to the operation which I have hitherto been able to find in Mr. Tait's writings in spite of pretty careful search. And I may as well say just here—that my knowledge of Mr. Tait's published writings is in no case second-hand, as he seems to imagine. I think far too highly of their distinguished author, and appreciate their positive and vigorous style too much, to allow any of them to pass me by unread.

Mr. Tait objects to my title of the Battey-Tait operation, because he and Dr. Battey do their respective operations for different reasons. That is quite true, but then the results of an operation are what it is judged by. It is doubtless very self-comforting for Mr. Tait to suggest that all the errors arising from removal of the ovaries are due to the ideas of his "distinguished friend Dr. Battey," while all the gains are due to his own practice of also removing the tubes. But that is not very convincing, and if we accept Mr. Tait's personal experience that he has done Battey's operation "five times and with such bad results" that he will "not continue the practice," I imagine it will be with the logical consequence of making the profession somewhat sceptical of the good results of "Tait's operation." In the meantime I must beg to retain the title I have adopted, if for no other cause than to emphasize the fact that there is so much diversity of reasoning underlying this operation.

The main points on which Mr. Tait and I are at issue concern the value of the procedure in cases of uterine disease, hemorrhage, and myomata. And this incidentally raises the question of the influence of the removal of the ovaries and tubes on menstruation.

Mr. Tait speaks with no uncertain sound on these points. While Dr. Battey proposed his operation of removal of the ovaries for the very purpose of bringing about the cessation of menstruation, Mr. Tait boldly declares that Battey's operation has little or no influence in this direction. On the other hand, he alleges that the Tubes are *fons et origo mali*, and that their removal alone five times out of six will stop the flow? How does he know that? In how many cases has he removed the tubes alone? On page 66 of his book on "Diseases of the Ovaries" (fourth edition, Wm.

Wood & Co., 1883) he says : "I always remove the ovaries along with the tubes, as without the ducts the glands are of course useless." Again, on page 327, in discussing this class of cases, he says : "In my own practice the conclusion is indicated that removal of the Fallopian tubes is more important than removal of the ovaries, and in by far the larger number of my cases that alone might have sufficed—indeed, in many it has done so." With the physiology underlying this practice I entirely dissent, and submit that it is radically unsound. Mr. Tait's main addition to our knowledge seems to me to consist in showing the only method of relieving a large class of sufferers from otherwise incurable salpingitis, but the incurable conditions in these cases are in nowise due to the functional importance of the tubes, but to their anatomical situation and conformation. In support of the very important functional rôle he ascribes to the tubes he has adduced no evidence. In his belief he apparently stands alone, for even though menstruation and ovulation were proven to be entirely distinct and independent functions—which is very far from being the case,—he would still have to bring forward any evidence that the tubes are the main factors in causing menstruation. The energy of Mr. Tait's statements on this whole question cannot obscure the fact that the whole burden of proof lies with him. He seems to me in this portion of his reply to be pleading like an advocate at the bar in defence of Tait's operation. That is a false mental attitude. I think on such an issue partisanship is not likely to carry much weight in overthrowing long-established scientific beliefs, and the impartial seeker for truth is tempted to exclaim, as a famous Englishman did after listening to a famous and dogmatic colleague : "I wish I was as cock-sure of any thing as 'he' is of every thing." The reasons I brought forward for holding the beneficial effects of the Battey-Tait operation as "not proven" for the cure of uterine myomata and hemorrhage, Mr. Tait makes no attempt to answer, and I need not therefore repeat them.

In discussing the small influence of the operation on the sexua appetite, I am glad to find that Mr. Tait and I so closely agree.

With regard to one point, Mr. Tait complains that I have done him an injustice in saying that he minimizes the risks of the operation. Now I was careful to point out that the wonderful results obtained by Mr. Tait went far to justify him personally in operating, but in his influence as a teacher I think he largely justifies my accusation. To go no further than his very reply in reference to my statement that "the more extensively the ovaries are matted up in the products of an old peritonitis, the greater will be the necessity for an operation," Mr. Tait says: "This is quite correct, but I cannot at all agree with Dr. Thallon when he further states that this suggests the unpleasant reflection that the greater danger is there of it being fatal." Now I would like to ask any fair-minded reader if it is unfair or unjust to say that Mr. Tait minimizes the danger of the operation when he says he cannot at all agree that extensive adhesions add to its danger?

With reference to the diagnosis of the contents of Fallopian cysts from the presence of columnar ciliated epithelium in their contents, the suggestion was based by Dr. Ferguson on the examination of a number of cases. The evidence will in due time be published, and in the meantime I can only say if the suggestion was "anticipated" by Mr. Tait, it certainly was not "exhausted" by him!

In conclusion I am glad to find Mr. Tait so entirely agrees with me in my statements regarding the large class of cases in which the operation is positively justifiable. In helping us to recognize this class of cases, especially those where incurable and painful disease of the tubes exists, Mr. Tait has done great and good service. But while willingly paying my tribute of admiration to the brilliancy of his practical results, I beg to enter my most earnest protest against what I esteem the false physiology, in spite of which those successes have been attained.

WILLIAM M. THALLON, M.D.

Brooklyn, *June* 22, 1884.

NEW BOOKS AND INSTRUMENTS.

Studies in Pathological Anatomy. By FRANCIS DELAFIELD, M.D., Professor of Pathology and Practical Medicine, College of Physicians and Surgeons, New York. Vol. II., Part 1.—"Broncho-Pneumonia." Plates i. to xii. New York : Wm. Wood & Co., November, 1883.

The first volume of Dr. Delafield's excellent "Studies" has been completed in 1880, and comprised the subjects of peritonitis, pleurisy, pneumonia, empyema, hydrothorax, bronchitis, tuberculosis, and phthisis ; illustrated with ninety-three plates. The work has received its due share of praise at home and abroad as one of the best productions of American pathology.

The fasciculus before us is a continuation of the above "Studies" and forms the first part of the second volume. The text consists of fifteen royal octavo pages, in large type, giving a concise description of the lesions comprised under the term *broncho-pneumonia*, and is illustrated by seven full-page photographs and five double-page lithographic plates ; all being reproductions of original photographs, and drawings from the author's skilled hand.

These illustrations are quite beautiful and well selected ; they give the work an exceptional value, and form its essential feature. Strictly speaking the work is an atlas of high artistic and scientific value, for advanced students and specialists, rather than a treatise of pathological anatomy. Some of the drawings are executed under enormous amplification (x 1,500, etc.) which, although quite instructive in giving the extreme histological details, are hardly accessible to the ordinary medical mortal, and less useful than the inviting pictures representing a moderate amplification, such as any student can see and compare with the specimen under the microscope. Fortunately, however the majority of Dr. Delafield's illustrations are of the latter class, and in proportion there are more of them in the fasciculus before us than in the first volume. An admirable feature are the author's photographs of the gross appearances or of pictures only slightly amplified. These are surely true to nature, are useful in conveying to the reader the exact and correct idea of the lesion under consideration.

The twelve illustrations referred to and the accompanying text give us, as stated, a very good and clear chapter of the author's views "on some of the forms of non-tubercular broncho-pneumonia."

Dr. Delafield appears to be an adherer to Virchow's view on the dual origin of phthisis ; he strictly distinguishes a non-tubercular from a tubercular form of broncho-pneumonia or phthisis. Admitting the difficulty in separating broncho-pneumonia from phthisis, he expresses his position and view on this question as follows :

" In both we find the same combination of peri-bronchitis and diffuse pneumonia ; the peri-bronchitic pneumonia characterized by the implication of the walls of the vesicles and the presence of permanent tissue in their cavities.

" In both the cellular infiltration of the walls of the bronchi may lead to their dilatation and to the formation of cavities.

" In both there are inflammatory changes involving the connective-tissue framework of the lung ; and when these changes have once commenced, there is the same tendency to a persistence and extension of the inflammatory process.

" In both we may have much the same clinical histories.

" But in broncho-pneumonia there are no tubercle tissue, no tubercle bacilli, no obliteration of blood-vessels, no areas of coagulation necrosis, and comparatively little cheesy degeneration of inflammatory products."

From croupous pneumonia the author distinguishes the lesion under consideration, thus :

In broncho-pneumonia " there is not merely an accumulation of inflammatory products in the cavities of the bronchi and air-vesicles, as in other forms of pneumonia, but also inflammatory changes in the walls of the bronchi and vesicles, so that the tissue of the lung is much more seriously changed, and changed in a way much more likely to remain permanent."

The author believes that in every pneumonia there is some bronchitis present : if this bronchitis is more prominent than the changes in the pulmonary tissue, or if the latter is affected only in limited areas around the bronchials, the lesion is then to be designated a *broncho-pneumonia.* The characteristic gross appearances of sections of such lungs are well shown in the author's photographs. Grayish peri-bronchitic nodules of various sizes and figures, often densely packed, are seen scattered throughout the parenchyma of the lungs, which otherwise may be merely congested or may be variously affected. Dr. Delafield states that the extension of the inflammation, from the affected bronchioles, is not to the air-vesicles which empty into that bronchus, but into those which are in contact with it. It is "as if a red-hot needle were thrust through the lung, making a tract of charred tissue around it. So each of these inflamed bronchi along its whole length is surrounded by a zone of inflamed air-vesicles."

The author distinguishes an acute form of broncho-pneumonia from a chronic or "persistent" form of this lesion. He describes and illustrates each form by well-studied typical cases observed among adults as well as children, but preëminently in the latter. Each case is accompanied by an autopsy record, microscopical studies, and instructive drawings or photographs of the lung lesions.

The acute form of broncho-pneumonia is identified with "capillary bronchitis" of the adult and the pneumonia of children which follows whooping-cough, measles, scarlatina, diphtheria, etc., but may also occur as an idiopathic affection.

Death may ensue within a few hours from the onset of the attack, or may last days or weeks. But recovery appears to occur quite commonly and perfectly in this affection.

Less frequently, the author says, a chronic inflammation of the interstitial tissue of the lung is developed and the child goes on to have a *persistent broncho-pneumonia.*

The attacks may repeat themselves year after year while the child grows up to adult life. Lungs gradually become consolidated, pleuræ thickened, never returning to normal condition, and "*the patient develops acute general miliary tuberculosis.*"

In all cases of persistent broncho-pneumonia the general conditions are given by the author as follows .

"(1) A chronic bronchitis with or without dilatation of the bronchi ; (2) the formation of new connective tissue in the walls of the bronchi and the air-vesicles, along the course of the blood-vessels, in the interlobular septa, and pulmonary pleuræ ; (3) obliteration of air-vesicles, and the production of epithelium and pus within them. The exact condition in each case depends upon the extent of lung involved."

The author lays further stress upon the emphysematous conditions and collapse of groups of air-vesicles or parts of lungs and upon occasional tuberculous inflammation of the bronchial glands as concurrent affections.

As a whole, the author's descriptions are clear, precise, and to the point. None but favorable comments can be justly made.

It is easy to recognize *acute* broncho-pneumonia as an independent affection. This affection in its typical form is, however, seldom seen upon the autopsy table, as the patients in a great majority of cases either get well or the disease has run a chronic course.

A closely analogous lesion, the induced or inhalation broncho-pneumonia, has of late repeatedly been studied in dogs and other animals and has thrown a good deal of light upon the lesion under consideration. If animals are forced to inhale irritative particles (sputum, pus, or powders of any kind) suspended in the air by means of a spray apparatus, or if the material is introduced through an opening in the trachea in the neck, an exquisite acute broncho-pneumonia ensues in every case. Similar results are often obtained also after paralysis of the epiglottis induced by section of both vagi in the neck, the saliva and particles of food being thus allowed to enter into the unprotected trachea and lungs.

Except in the latter case, the animals as a rule recover within the second month after the operation (even after sputum inhalations), and if an autopsy is made of an animal killed after the lapse of two months the lungs are seen perfectly normal. When, however, the animal is examined within the first month there is seen an *acute* broncho-pneumonia not distinguishable from that described in man by Dr. Delafield. Throughout the parenchyma of the lungs are seen small nodes, of irregular sizes and outlines, resembling tubercles.

Under the microscope these nodules show to consist of mere unorganized collection of cells, often in a state of retrograde change and mixed with mucus and débris ; this exudate being limited to the lumina of the bronchials and their pertaining groups of air-vesicles (the acini) which they fill. On section these artificial boundaries give rise to the appearance of nodes. Very often epithelial cells and mucus from the bronchi have been seen intermingled with the purulent contents within the air-vesicles ; this suggests that the exudate contained in the latter may have been partly aspired from the bronchi (during forced breathing) and was lodged in the air-vesicles as foreign material. This appears to be also the case in acute broncho-pneumonia in man. In fatal cases of acute broncho-pneumonia in animals as well as in man abscesses have been met with.

Chronic broncho-pneumonia, however, must be classed with the tuberculosis lesions in accordance with our present knowledge of what constitutes tubercle ; a knowledge to which Dr. Delafield himself has so much contributed in his "Studies."

From the standpoint of a unitory origin of phthisis it is difficult to conceive a definite distinction between *chronic non-tubercular* and *tubercular broncho-pneumonia*, first on account of the too common concurrence of miliary tuberculosis, and further for other reasons stated by Dr. Delafield himself.

As a really refreshing feature of the author's admirable "Studies" must be mentioned yet his sound and unbiassed reference to the tubercle-bacillus question. [H. F. F.]

Psychiatrie : Klinik der Erkrankungen des Vorderhirnes, begründet auf dessen Bau, Leistungen und Ernährung. Von Dr. THEODOR MEYNERT, Professor der Nervenkrankheiten und Vorstand der Psychiatrischen Klinik in Wien. Erste Hälfte. Wien, 1884. Wilhelm Braumüller.

In an article published in 1878, Professor Meynert incidentally refers to his treatise on psychiatry which he was then preparing for the press. More than six years have since elapsed, and the first half of that work has but just been presented to the medical public. The delay in its publication can not be attributed solely to the author's avowed dislike of book-making; in part it is undoubtedly due to the careful manner in which Prof. Meynert has sifted his own thoughts, and the facts furnished by others, before putting them to print. Having a distinct message to impart, he knew that, whether published a year or two earlier or later, the originality and worth of his views would not be disputed by others.

To all medical men, and particularly to those interested in the study of the brain, Meynert's name is well known. His anatomical researches rank with those of Clarke, Burdach, and Stilling. Our present knowledge of the relation of the subcortical ganglia to the hemispheres, of the minute anatomy of the cortex, of the tegmentum, and of the projection-systems, is based largely upon the investigations of Meynert. No wonder, that Wernicke, an excellent judge in such matters, should call him "the greatest brain-anatomist that ever lived" !

Of late years Meynert has put his extraordinary knowledge of the structure of the brain to the best possible use—to the study of the physiological and morbid conditions of that organ. At various times and in various pamphlets he has indicated the nature of the problems he has endeavored to solve. This attempt to strike out on a new line of research in the study of the origin and nature of mental diseases lends special importance to the work before us. The title-page tells its story : Mental diseases are declared to be diseases of the fore-brain, due to disorders in the structure, functions, and nutrition of that part of the encephalon. Former authors, and among them the greatest names in this domain of medical science on both sides of the Atlantic, have studied carefully the clinical manifestations of mental disease, but have never ventured an explanation of the various forms of insanity based upon anatomical and physiological data. A recent writer on this subject condemns every effort to lift the curtain from the mysteries of mind, as premature and imprudent. Meynert is of a different opinion. He thinks there is no need of leaving it to posterity to make deductions from anatomical and physiological discoveries which we have made. New facts should be utilized at once. Mistakes are inevitable, but succeeding investigators will profit by them.

In the very first chapter, on the anatomy of the brain, the author shows his thorough mastery of the subject ; in this section those structures are mapped out and thoroughly explained which have an important bearing upon the study of mental diseases. Those who expect this part of the treatise to take the place of a text-book on cerebral anatomy will be sorely disappointed. Among recent authors, Wernicke, Schwalbe, Luys, and Meynert himself (in Stricker's " Handbook of Histology ") have given more detailed accounts of the minute structure of the brain. Instead of burdening the reader's mind with a vast array of facts, Meynert prefers, in this instance, to have him conceive the brain as a unit, and to have him understand the relative arrangement of its parts. Sufficient embryological data are given to show the development of the adult brain from the primary medullary tube, and more especially the growth of the fore-brain from the lateral appendages of the primary anterior vesicle.

Whether we consider the cerebral structure from an anatomical, physiological, or biological point of view, we must, according to Meynert, distinguish between the cortex and the sub-cortical ganglia ; we must remember that the cortex is the one surface upon which all the impressions from the body and the outer world are registered, and that the sub-cortical ganglia are way stations, as it were, through which the peripheral nerve-tracts pass on their journey to the cortex.

The division of these nerve-tracts into projection-systems is too well known to be more than alluded to here. The other (intra-cortical) system of fibres—the association-system—plays an important rôle in the physiological and later sections of this book. Professor Meynert has devoted great care to the proper elucidation of these fibres. Lying entirely within the cortex, and forming closely interlacing groups, they can be most clearly demonstrated by having recourse to the old method of detaching the various bundles of fibres from their proper connections, without creating artificial divisions. Forel has been loudest in his objections to this "detaching" method (Abfaserungsmethode). It may prove a dangerous weapon in the hands of an amateur anatomist, but not when practised by Burdach or Meynert. The author's preparations, of which excellent drawings are given in the book, convince one of the truth of what they are intended to express.

Meynert has deprived cerebral anatomy of much of the mystery which once attended it.

Of great interest in the chapter still under consideration are the brief allusions to the cause of variation of cerebral structure among the different species of animals. Thus he clearly shows that the greater or lesser development of the fore-brain is the controlling factor in shaping the entire cerebral mass ; that upon such variations in development depend the relative size of the *pes pedunculi*, of the pons and the pyramidal tracts, and indirectly the appearance or (apparent) disappearance of the *corpus trapezoides* and olivary body in animals and man.

Excellent as this chapter is in all other respects, we regret that it was not revised shortly before the appearance of the first half of this work, for during the last few years Forel, Schwalbe, v. Monakow, and particularly Roller, have added greatly to our knowledge of the minute anatomy of the brain. It is barely possible, however, that Meynert will refer to the conclusions of these investigators in an appendix which he promises to issue with the second half of the volume.

From the chapter succeeding the one just reviewed, the student of psychology, as well as the medical man, may reap a rich harvest. As in the anatomical part, so here the intimate relations between the cortex and the sub-cortical ganglion is forced upon our attention. There is a *functional* relation corresponding in importance to the *anatomical* relation between these parts. The normal functions of the *cortex* are considered first. Once having accepted Bell's law of the centripetal and centrifugal conducting-power of the posterior and anterior nerve-roots respectively, the author shows that we need merely postulate the sensitiveness of the cerebral nerve-cell in order to gain an insight into the cortex as the organ of intelligence. Allowing the cortical surface of the child's brain to be a *tabula rasa*, let us suppose that it (the child) meets for the first time a bleating lamb. Through the various parts of the projection-system two impressions are carried to the cortex : a visual image of the lamb, and an auditory image of the sound it produced. These images, we have good reason to believe, will be stored in different parts of the cortical surface ; as there is no irradiation (practically) in the gray substance of the cortex, the cells, harboring these impressions, would be anatomically and functionally separate but for the presence of the association fibres connecting them. Such an association, once formed, is a permanent one, and one of these images cannot be excited without recalling the memory of the other. The cerebral cortex is practically a storehouse of such visual, auditory, and other images. An association group may comprise any number of distinct images or memories. Thus, as the author puts it, a child that has burned its fingers in a flame will add to the visual memory of the flame and to the memory of the painful tactile sensation, the memory of the reflex effort to withdraw the hand from the flame. Here the memory of muscular innervation comes into play ; and later on the author shows that voluntary movements of which a higher animal that lacks cerebral hemispheres is incapable, are simply effected by re-exciting the impressions registered in the cortex of the previous innervations of certain groups of muscles from the sub-cortical centres. The sum of all such impressions constitutes what we call intelligence. These impressions are scattered over different parts of the entire brain, 'and hence intelligence must be said to result from the activity of the entire cortex, and not from any part of it. To the development of logical reasoning from the intensity and frequent excitation of definitely associated groups of impressions we can only allude in passing. The explanation furnished of aggressive and repulsive movements has a more direct bearing upon mental diseases.

These are intimately connected with pleasurable and painful emotions. Relying upon the experiments of Schiff and Goltz, Meynert insists that in the case of the decapitated frog, pinching the leg not only causes reflex movements, but that the greater the irritation the greater the resistance offered in the gray substance of the spinal cord to the conduction of the irritant impulses, and that consciousness, when present, recognizes this inhibition as a painful sensation. Furthermore, it has been shown that repulsive movements are attended by a contraction of the blood-vessels of the spinal-cord segment through which the sensory irritation passes ; this in turn causes what the author calls the dyspnœtic phase of the nutrition of the nervous elements. Aggressive movements, on the other hand, are produced by very slight friction (Goltz's croak experiment), are accompanied by pleasurable sensations, due to the unrestricted passage of nerve force, by a functional hyperæmia, an apnœtic phase of nutrition, and so on. Pushing still farther in this direction, the author shows that an actual external irritant impulse is not needed to start this complicated mechanism, but that the mere memory of a former pain may be sufficient to cause a repulsive movement and all its concomitant states. Let those to whom these views appear fanciful look up the full arguments in the book itself before passing judgment upon them. This chapter and the following, on the nutrition of the brain, are full of interesting suggestions to which even numerous extracts would do but scant justice.

In the short chapter on the clinical manifestations of prosencephalic disease, the first practical application is made of anatomical and physiological principles as laid down in the preceding sections of this volume. A few difficulties meeting us at the outset are frankly stated : First—Symptoms due to sub-cortical irritation may screen cortical *lesion-symptoms* (Ausfallserscheinungen). Secondly—Symptoms equal in severity to true lesion-symptoms may be due to irregularities in the nutrition of an otherwise healthy part.

It will be a surprise to many to find predisposition to mental disease put down among the lowest forms of anatomical disorders. I question very much whether in all cases of mental troubles, and particularly in those with a strong *inherited* tendency to disease, it will be possible to assign an anatomical reason ; but Meynert ascribes the predisposition either to some injury to the head during parturition and consequent malformation of the skull, or to the constitutional structural anomalies attendant upon anæmia and other such causes. Whether successful or not, the attempt to define the exact nature of predisposition to disease is a creditable one, and deserves to be extended to other domains of medical science.

In considering functional disorders we must again discriminate between the cortex and sub-cortical centres. The first effect of a change toward defective nutrition of the cortex is a loss of cortical inhibition ; as soon as this inhibitory force is removed the play of the sub-cortical centres begins ; hallucinations and deliria are the result. *Irritation* of the sub-cortical centres may bring about the same result, while a *weakening* of the sub-cortical functions produces an increase of cortical power. Meynert explains this in the following way : Supposing that general anæmia of the brain has produced exhaustion of the hemispheres and of the sub-cortical vaso-motor centres, then a paretic condition of the arterial muscular walls will ensue, and the result is hyperæmia of the cortex. As long as the condition lasts we get a full complement of maniacal symptoms, viz. : pleasurable mood, an abundance of loosely associated ideas and innumerable motor impulses. By finally causing an arterial hyperæmia in the vaso-motor centre itself, this condition works out its own salvation ; the vessels of the cortext contract normally again, and the maniacal symptoms disappear. If this process (of vaso-motor irritation) is carried to the extreme the contraction of the cerebral vessels will become more intense, and all the symptoms of a melancholic mood will be developed.

From the explanation given of normal cortical functions, it is evident that irritation of the cortex could never produce any thing but abnormalities of thought—insane ideas—and pathological emotions ; but the latter have been shown to be mere intensified perceptions. We have already so far exceeded the limits of a review that we can not now dwell upon the manner in which, as the author shows, insane ideas and pathological emotions react upon each other ; nor do more than quote the opinion of Meynert (not peculiar to him, however,) that insane ideas are never newly formed ; that the insane subject (with defective cortical inhibition) simply reproduces such primitive ideas as a child possesses that has no proper appreciation of the relation of its own individuality to the surrounding world. The exaggerated or otherwise defective notions of the normal child are limited and corrected by experience.

The quintessence of Prof. Meynert's views is embodied in his *natural* system of classification of diseases of the fore-brain. It would be an injustice to the author to criticize or even to discuss this classification until after the publication of the clinical portion of the work. If the second instalment of this book will redeem the promises of the part we have reviewed, Prof. Meynert's treatise will form a landmark in psychiatrical literature.

For the benefit of those who will be puzzled by the labyrinthic sentences of the original, we add that an English translation of the book will soon be published by Messrs. G. P. Putnam's Sons, of this city, with the coöperation of a London firm. [B. S.]

Leçons sur les Maladies Mentales. Par le docteur B. Ball, Professeur à la Faculté de Médecine de Paris. Asselin et Cie. Paris, 1883, pp. 884.

The years 1883–84 will remain remarkable for the appearance of an extraordinary number of treatises and text-books on mental diseases—a flood apparently out of all proportion to the demand, and certainly not justified by any special advance in our knowledge of cerebral physiology or pathology. Besides the work before us, we may mention, as a partial list of these new books or editions, Krafft-Ebing's "Lehrbuch der Psychiatrie," 2d edition, 1883 ; Spitzka : "Insanity ; its Classification, Diagnosis, and Treatment," New York, 1883 ; Hammond : "A Treatise on Insanity," New York, 1883 ; Mann : "A Manual of Psychological Medicine," Phila., 1883 ; Hamilton : "Types of Insanity," New York, 1883 ; Neumann : "Leitfaden der Psychiatrie," Breslau, 1883 ; Clouston : "Clinical Lectures on Mental Diseases," London, 1883 (American edition, Phila., 1884) ; and Meynert : "Klinik der Erkrankungen des Vorderhirns," Bd. I., Wien, 1884. There are, doubtless, others, but this partial list is surely enough to appall the would-be thorough student of psychiatrical literature.

The work before us is, perhaps, the most purely clinical of all ; it is scarcely pathological, and not at all psychological. It aims, in beautifully clear, concise, often epigrammatic French, to delineate insanity as the physician will see it. Shall we say that it is even more clinical than a work written by a man brought up in the strict asylum hierarchy would be ? This would not be an exaggeration, and would find its explanation and justification in the well-known fact, that Prof. Ball was a master in ordinary clinical medicine before he began treating psychiatry. The scientific alienist may find great gaps in his work, but the practising physician will turn to it with ever-renewing pleasure and profit. The work is divided into forty-five lessons or chapters, and is, in reality, a double book, for, up to chapter xxi., the author describes morbid mental states as classically designated by the French school, while beyond this point he takes up the systematic description of the forms of insanity according to a classification of his own, which is (and the author admits it) based on the same etiological and pathological principles as that of Morel. Ball groups insanities as follows :

1. Vesanic or essential .	Circular insanity, Monomanias.
2. Neuropathic . .	Hysterical, Epileptic, Choreic, etc.
3. Diathetic . . .	Gouty, Rheumatic, Tubercular, Cancerous, Anæmic, etc.
4. Sympathetic . .	Genital, Cardiac, Gastro-intestinal, Pulmonary, etc.
5. Toxic . . .	Alcoholic, Saturnine, Morphinic, etc.
6. Organic, or cerebro-spinal	General paralysis, Aphasia, Acute delirium, Dementia with hemiplegia, etc.
7. Congenital . .	Idiocy, Imbecility, Cretinism.

The author does not attach overmuch value to this classification, but claims for it that it will meet the want of the clinician.

In chapters xvi.–xviii. the causes of insanity are well discussed. The great importance of inheritance, when rightly conceived, as a part of evolution, is broadly and strongly sketched. Other causes are classified as general and exciting. Among these injury to the head takes rank as most important. The efficacy of moral causes, especially that of the struggle for existence in a complex civilized state, acting on predisposed organizations, is admitted. General paralysis may result from injury to the head. In another place (chapter xxviii.) the author agrees with Maudsley in thinking that this disease may be caused by conjugal sexual excesses.

The pseudo-general paralyses from lead, from alcohol, from syphilis, and from pellagra, receive attention.

Alcoholism and its numerous psychic manifestations receive what might at first sight seem an undue share of attention ; it occupies chapters xxx. to xxxiii. But if one considers the extraordinary increase of alcoholic excesses in France (and other parts of Europe) in the last twenty years, and its ever-increasing influence as a direct and indirect factor in insanity and race-deterioration, this is justified. Dipsomania is recognized as a morbid state.

General paralysis occupies chapters xxxv. to xlii., inclusive, and is very well treated.

Dr. Ball's therapy of insanity is not encouraging. He has no optimism, and is evidently much influenced by his faith in the degenerative nature of many forms of insanity. Baths, warm and prolonged, and irritant baths, he favors. Opium and morphia are seldom beneficial. He recommends the bromides in melancholia, in epileptic insanity, and in insanity based upon hysterical states. Hyoscyamia is not mentioned, and chloral is said to be the best of all sedatives.

Seclusion is strongly advocated, either at home (imitating asylum conditions) or in institutions.

Non-restraint finds no favor in our author's eyes. He has evidently not seen for himself how perfectly the system works in Great Britain, Germany, and Switzerland, though a week's absence from Paris would have enabled him to do this. He would mechanically restrain suicides and destructive patients. He justly condemns giving chloral and morphia simply to insure quiescence, and probably overestimates the frequency of this practice in asylums.

The work will well repay study, though it is perhaps too bulky for translation. [E. C. S.]

"B"

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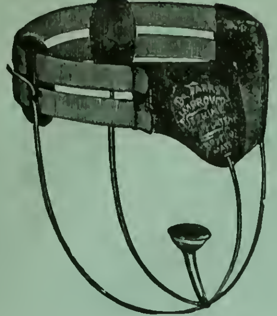

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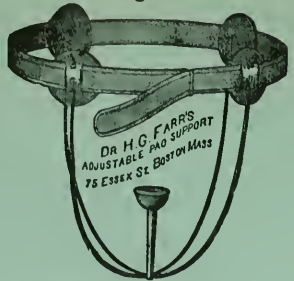

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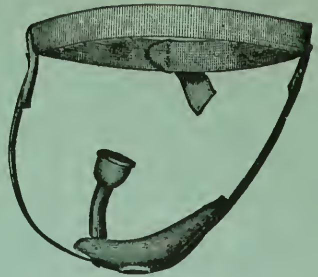

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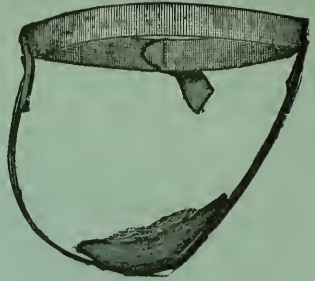

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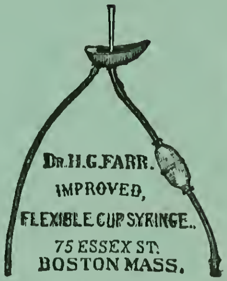

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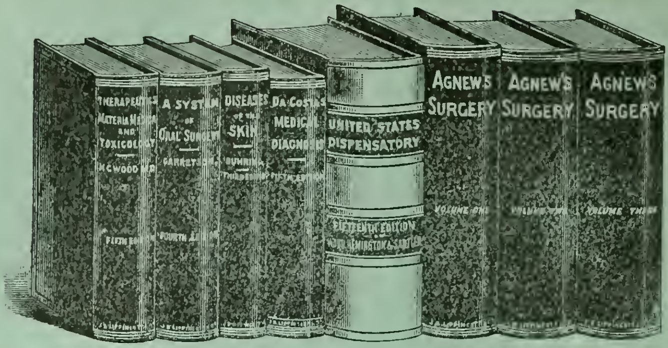

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THE
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OF
OPHTHALMOLOGY

EDITED BY

ADOLF ALT, M.D.,

St. Louis, Mo.,

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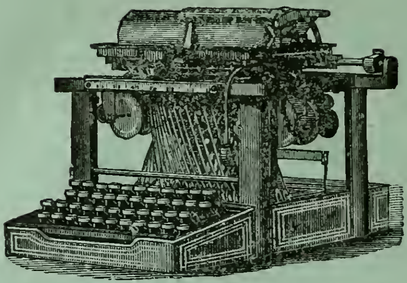

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METRIC TABLE.

As the Metric System *will be used in this Journal, the* ***following Tables****, for reference, are given for those not yet familiar with the system :*

METRIC MEASURES OF LENGTH.

1 Millimetre	0.001	= .039	inches
1 Centimetre	0.01	= .393	"
1 Decimetre	0.1	= 3.937	"
1 METRE	1.	=39.370	"
1 Kilometre	1000.	= .62	miles

METRIC WEIGHTS.

1 Milligram . . .	0.001	=$\frac{1}{64}$	gr.
1 Centigram . .	0.01	= $\frac{1}{6}$	"
1 Decigram . . .	0.1	=$1\frac{1}{2}$	"
1 GRAM	1.	=15.432	
1 Kilogram . .	1000.	=2.7	lb.

APPROXIMATE EQUIVALENTS.

1 ♏ or 1 gr.	= .06	grams
1 f ʒ or 1 ʒ	= 4.	"
1 f ℥	=30.	"
1 ℥	=31.	"
1 f ℥ Glycerine . .	=37.	"
1 f ℥ Syrups . . .	=40.	"

TEMPERATURE.

37° Cent.	98°.6	Fahr.
38° "	100°.4	"
39° "	102°.2	"
40° "	104°.	"
41° "	105°.8	"

1° C.=1°.8 F. Multiply C. by 1.8 add 32=F.

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